International Clinical and Medical Case Reports Journal
Case Report (ISSN: 2832-5788)

Xylazine And Brugada Syndrome: Unveiling The Hidden Risk

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Citation: *Vidhi Mehta, Shrikanth Sampath, Shanjeev Kumar Rajeshkumar Chitra, Sruthi Sekaran, Mahvish Renzu. Xylazine And Brugada Syndrome: Unveiling The Hidden Risk. Int Clinc Med Case Rep Jour. 2024;3(3):1-2.*

Received Date: 05 March, 2024**; Accepted Date:** 28 March, 2024**; Published Date:** 30 March, 2024

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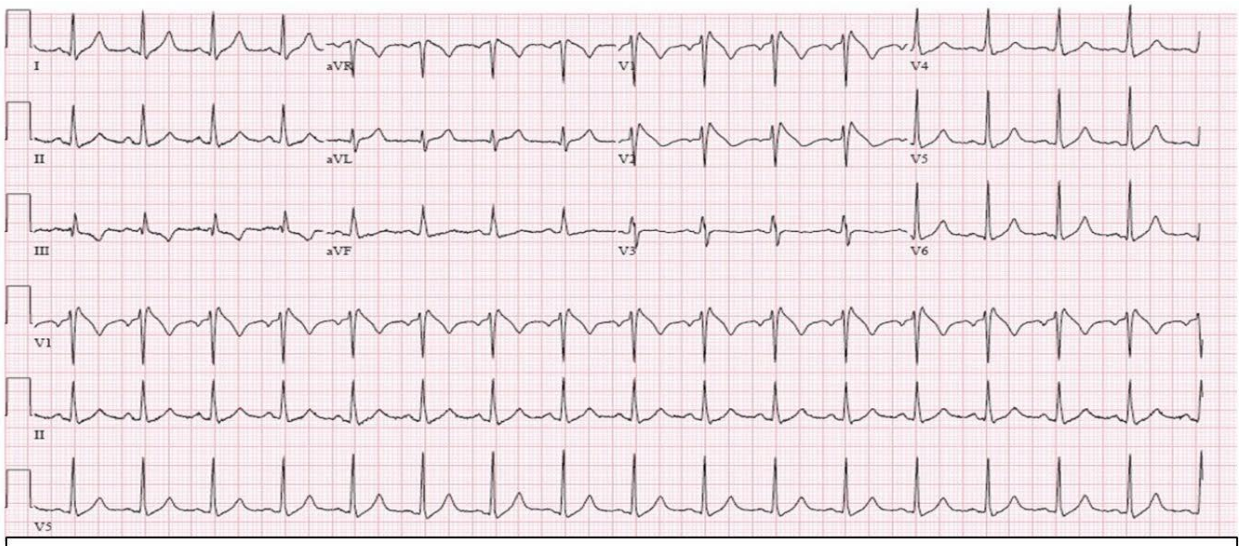
INTRODUCTION

Brugada syndrome is a rare, but potentially life-threatening heart condition, characterized by abnormal electrical activity in the heart, leading to irregular heart rhythms. It is often inherited and may not show any symptoms until triggered by certain factors like fever/ infection, medications, etc. In this case we bring forward one such unique presentation of brugada syndrome which was precipitated by xylazine use.

CASE DESCRIPTION

A 27-year-old male with a history of anxiety and intravenous opioid use was admitted after being found unresponsive and hypoglycemic by EMS. He endorsed using fentanyl with xylazine before passing out and was tachycardic, hypotensive with anion gap acidosis, transaminitis, and elevated troponins peaking at 788 ng/L. UDS positive for fentanyl and cannabinoids. Family history of sudden cardiac death (SCD) in uncle with initial ECG seen in Figure 1.0.

Echocardiogram demonstrated a left ventricular ejection fraction of 50% and a dilated right atrium with increased pressure (15 mmHg). CT chest showed possible pneumonia and subsegmental pulmonary emboli managed with antibiotics and anticoagulation. Repeat EKG on day 3 showed a similar Brugada sign in V1 but showed Brugada type 2 saddleback-shaped ST elevation in V2. Patient left against medical advice before planned electrophysiological evaluation and intervention.



EKG on day 1: Right bundle branch block, coved ST elevations in V1 and V2 with inverted T waves concerning for Brugada syndrome type I.

DISCUSSION

Xylazine, an alpha-2 adrenergic agonist, is used in veterinary practice as a sedative and analgesic. Its exposure to humans has increased due to recreational drug use in the form of illicit combinations or adulterated substances. Brugada syndrome is an inherited sodium channelopathy characterized by distinct EKG abnormalities including coved ST elevation and T wave inversion in right precordial leads (V1 to V3) predisposing to lethal arrhythmias. It has been recognized as a potential cause of SCD, especially in young individuals with structurally normal hearts. While the exact mechanisms are ambiguous, xylazine is known to inhibit cardiac sodium channels from animal studies. This may have unmasked Brugada type 1 pattern in our patient with non-type 1 syndrome seen in the subsequent EKG, thereby increasing the risk of malignant arrhythmias. Further investigation is warranted to better understand the cardiotoxic e!ects of xylazine. Consequently, attention and precautionary measures are needed to protect individuals at risk.

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