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A Rare Case of Bupivacaine-Induced Cardiac Toxicity Presenting as Acute Coronary Syndrome in Emergency Department

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ABSTRACT/ INTRODUCTION

Bupivacaine is a long-acting, local anaesthetic (LA) agent which is widely used for cutaneous infiltration, intra-articular injection, peripheral nerve blocks, epidural and spinal anaesthesia[1]. Most fatal side effects of local anaesthetics are due to the involvement of cardiovascular and central nervous system[2]. LA toxicity occurs either due to overdose of these agents or accidental intravascular injections which usually involve cardiovascular & nervous systems simultaneously. Cardiovascular side effects are mainly due to Myocardial conduction depression & negative inotropic action. Bupivacaine produces a concentration-related depression of intra-atrial, A-V nodal, intraventricular conduction and myocardial contractility owing to a fast sodium channel blocking in both nerve and cardiac tissue[3-5]. However, there has been no report concerning bupivacaine-induced myocardial injury presenting to the Emergency Department (ED) as acute coronary syndrome. We present a case mimicking acute coronary syndrome non-ST segment elevation myocardial infarction (NSTEMI), which was diagnosed as a bupivacaine-induced cardiac injury without CNS toxicity.

Keywords: Bupivacaine; Cardiac Toxicity; Coronary Syndrome; Emergency Department

CASE PRESENTATION

A 24-year-old young male (Non-smoker, Non-alcoholic & Non-Drug abuser) patient presented to our emergency department with the chief complaints of chest pain& dyspnoea since 2 hours. This patient was referred to our hospital where he was scheduled for orchidopexy in the morning because of trauma to scrotal region 1 year back. He had normal cardiac, respiratory, liver and renal functions with normal laboratory findings on preoperative examination. Vital signs were normal during the operation. Spinal anaesthesia was given with a 25G Quinche spinal needle at the L3-4 interspace in the sitting position. Clear and free-flowing cerebrospinal fluid was observed, and

heavy bupivacaine 15 mg was administered. After 5 mins of Bupivacaine injection, he started complaining of chest discomfort & severe dyspnoea. Simultaneously, he developed tachycardia and hypertension. He had to be taken off the operating table& his operation was cancelled. Injection Esmolol 20 mg IV was given there, and the patient was monitored in ICU for an hour. After initial stabilisation, the patient remained symptomatic so he was referred to our centre for further management.

When he arrived to our emergency department his chief complaints were chest pain & mild dyspnoea. Chest pain was left-sided, non-radiating in nature. There were no other complaints. His Pulse was 132/min, Blood Pressure was 114/80 mm of Hg, Respiratory rate was 24/min,SpO2 was 99% on room air & body temperature of 98.6 F. His chest was clear & other systemic examination was unremarkable. His Electrocardiogram (ECG) was done on arrival (Figure1) which showed Sinus Rhythm with ST segment depression with T wave inversion in Lead V5-V6 & Lead I, II, III, aVF. His chest radiograph was normal. Screening Echocardiograph which was done in ED showed Mild global hypokinesia with LVEF 45-50%. His Trop I was 1.05 ng/mL (reference range: < 0.2 ng/mL).Other laboratory findings were normal except mild leucocytosis (Total Leucocyte Count 13400/mm^3).On the basis of her symptoms, signs, laboratory and imaging results, Acute Coronary Syndrome Non-ST elevation Myocardial Infraction was made in ED. Tablet Asprin 325 mg orally, Tablet Clopidogrel 300 mg orally, Tablet Atorvastatin 80 mg orally & Injection Heparin 4000 IU intravenously was given to him in ED. Further, he was admitted to the Cardiac care unit. After admission, his Coronary angiography was done which showed normal coronaries (Figure 2). He was treated symptomatically along with hyperpolarization-activated cyclic nucleotide-gated (HCN) channel blockers Ivabradine 5 mg orally. After the next day of admission, his symptoms started improving. His follow-up ECHO was done before discharge which was normal. Also, at the time of discharge ECG & Cardiac Enzyme was done, ECG showed normal sinus rhythm (Figure 3) while his cardiac markers were within normal limit. Subsequently, he got discharged after 2 days of admission without any complications.

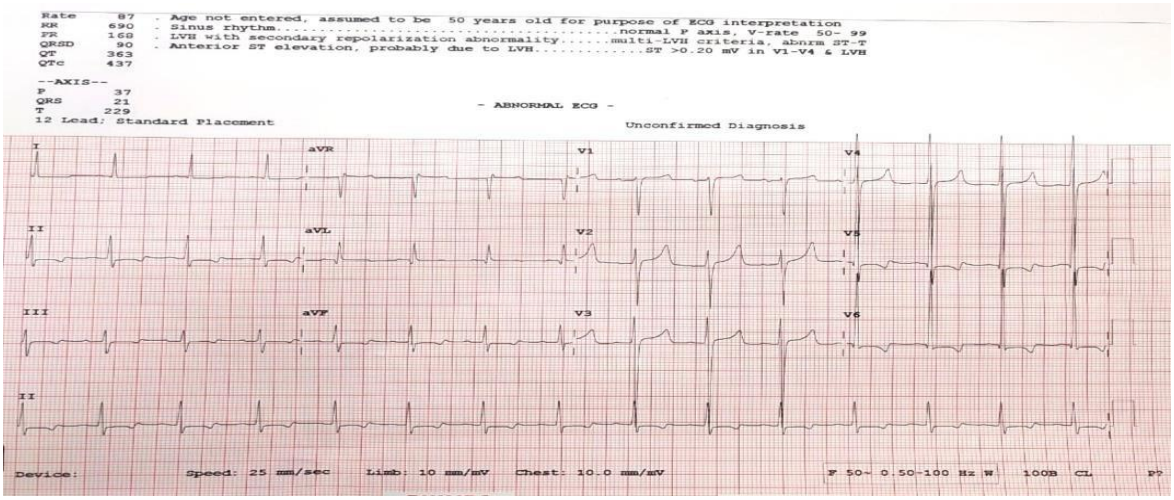

Figure 1: Initial ECG showing Sinus Rhythm with ST-segment depression with T wave inversion in V5-V6 & Lead I, II, III, aVF

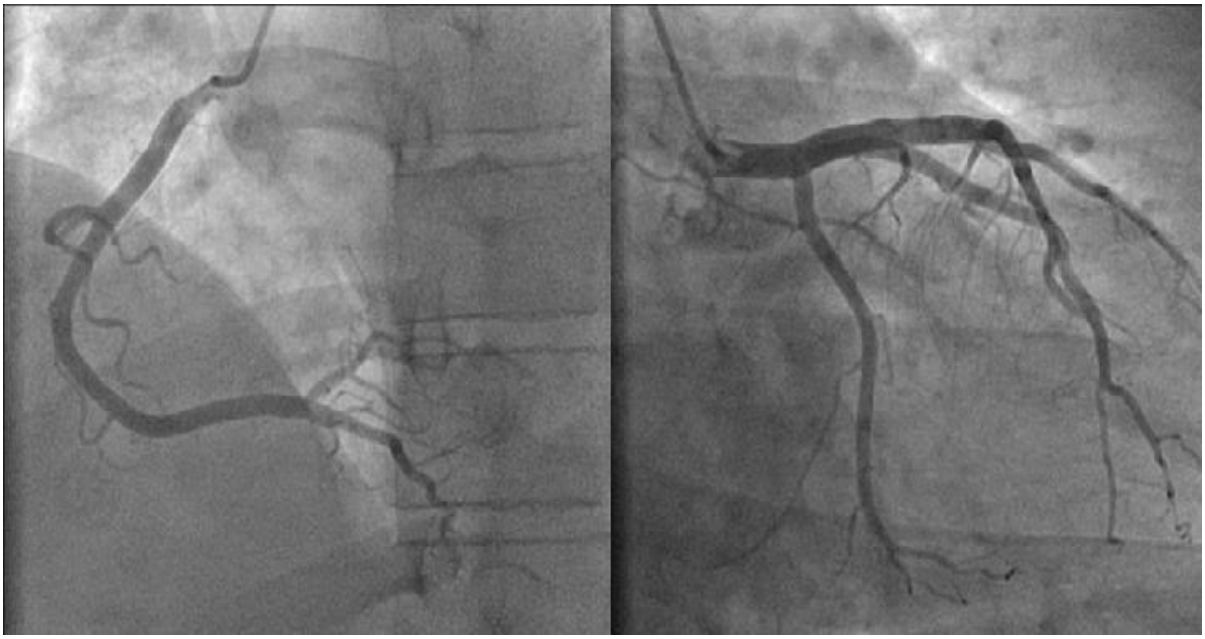

Figure 2: Coronary Angiogram showing normal coronaries

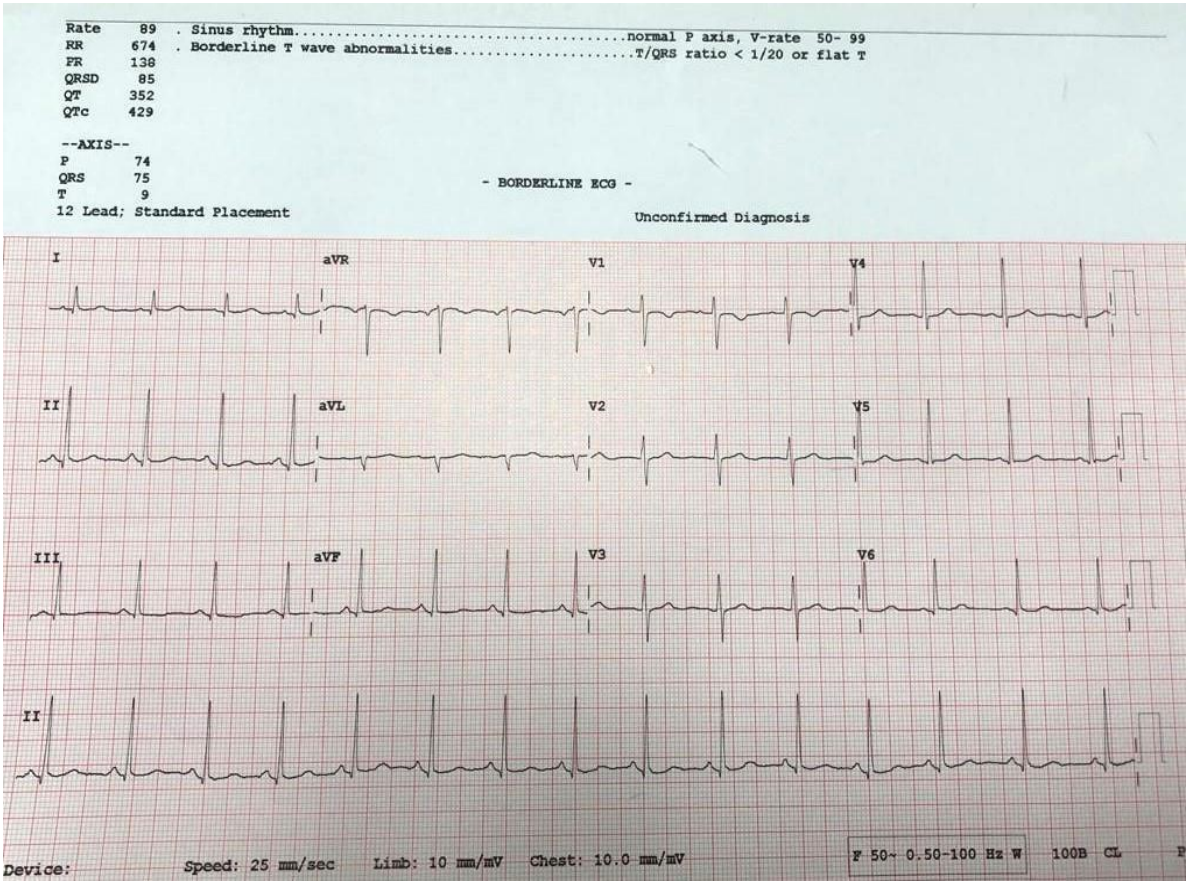

Figure 3: ECG before discharge showing normal findings

DISCUSSION

Bupivacaine is widely used as a long-acting local anaesthetic agent; however, acute and fatal side effects of local anaesthetics usually occur simultaneously after an overdose of local anaesthetics or accidental intravascular injections[6].

Bupivacaine side effects are related to CNS and adverse cardiovascular effects. CNS side effects usually occur before the cardiovascular signs and symptoms. Cardiac toxicity is one of the adverse effects of bupivacaine; it can present as cardiac depression, ventricular tachycardia, asystole, and/or electromechanical dissociation. This is due to the blocking of sodium channels in the cardiovascular system[3,4]. Bupivacaine decreases the maximum diastolic potential and action potential amplitude in myocardial tissue and prolongs the ratio of the effective refractory period to action potential duration[3,7]. All this produces a depression of myocardial conduction and negative inotropic action.

In our case, when the patient presented to ED, he had typical features of Acute coronary syndrome like chest pain, dyspnoea & sweating, and elevated Troponin level. We made our initial diagnosis as Acute Coronary Syndrome Non-ST elevation Myocardial Infarction but there were several contradictory features here, as he was a young male without any positive family history of cardiac disease & there were no risk factors. There was mild global hypokinesia on ECHO findings and symptoms started after receiving injection bupivacaine. Initially, we kept our differential diagnosis as Stress cardiomyopathy, Viral Myocarditis, Anaemia, and Coronary Vasospasm.

Firstly, Stress cardiomyopathy was ruled out as there was no history of any emotional stress which is the main precipitating cause of cardiac symptoms. Stress cardiomyopathy shows a unique pattern of left ventricular dysfunction characterised by apical and mid-ventricular contractile abnormalities with sparing of the basal segments on ECHO. There were no such features in our case on ECHO[8].

Secondly, viral myocarditis was ruled out because he did not have any Fever, fatigue or myalgia before or after bupivacaine administration. Furthermore, there was no usual tachycardia in viral myocarditis[9]. In addition, all his laboratory findings were normal. Anaemia also was also ruled out as his lab reports were normal.

There was mild global hypokinesia on ECHO but his coronaries were normal on angiography so coronary vasospasm was ruled out later on. Finally, after ruling out other differentials diagnosis, we confirmed it was Bupivacaine induced cardiotoxicity only.

In a review of the literature, some reports show adverse effects of bupivacaine. Scott DB et al. reported depression of conductivity and contractility after intravenous injections of bupivacaine in 12 healthy volunteers[10]. Coven et al. reported two cases with an accelerated idioventricular rhythm during spinal anaesthesia using bupivacaine for a caesarean section[11]. Furthermore, Cotileas et al. reported a case, in which bupivacaine-induced myocardial depression and pulmonary edema were described in a healthy young woman with epidural anaesthesia for lipoanarrofisis, although she had normal cardiac enzyme levels[12].A case similar to our case were reported by Ryu

HY in 2007 where patient had cardiogenic shock along with features of Myocardial infraction after bupivacaine injection[13].Another case which was reported by Jin Yong Parket al. where patient had fatal peripartum cardiomyopathy after bupivacaine injection for elective caesarian section[14].

CONCLUSION

Bupivacaine can cause a variety of cardiac toxicity symptoms without any neurological symptoms. Among those, few may be fatal arrhythmias while in some case it may be reversible myocardial depression. Through history & awareness by the Emergency physician can help in timely diagnosis & prompt management of any cardiotoxicity caused by Local Anaesthetics in the ED.

REFERENCES

1. Cordell CL, Schubukegel T, Light TR, Ahmad F. Lipid infusion Rescue for Bupivacaine-induced Cardiac arrest After Axillary Block. J Hand Surg Am. 2010; 35:144-146.
2. Knudsen K, Beckman Suurkula K, Blomberg S, Sjovall J, Edvardsson N. Central nervous and cardiovascular effects of i.v. infusions of ropivacaine, bupivacaine and placebo in volunteers. Br J Anaesth. 1997;78: 507–514.
3. Arlock P. Actions of three local anaesthetics: lidocaine, bupivacaine and ropivacaine on guinea pig papillary muscle sodium channels (Vmax) PharmacolToxicol. 1988;63:96–104.
4. Clarkson CW, Hondeghem LM. Mechanism for bupivacaine depression of cardiac conduction: fast block of sodium channels during the action potential with slow recovery from block during diastole. Anesthesiology. 1985;62: 396–405.
5. Heavner JE. Cardiac toxicity of local anesthetics in the intact isolated heart model: a review. Reg Anesth Pain Med. 2002;27: 545–555.
6. Neal JM, Bernards CM, Butterworth JF, Di Gregorio G, Drasner K, Hejtmanek MR. ASRA practice advisory on local anesthetic systemic toxicity. Reg Anesth Pain Med. 2010;35: 152-161.
7. Moller R, Covino BG. Cardiac electrophysiologic properties of bupivacaine and lidocaine compared with those of ropivacaine, a new amide local anesthetic. Anesthesiology. 1990;72:322–329.
8. Wittstein IS, Thiemann DR, Lima JA, Baughman KL, Schulman SP, Gerstenblith G. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med. 2005;352:539–548.
9. Matsouka H, Hamada M, Honda T, Kawakami H, Abe M, Shigematsu Y. Evaluation of acute myocarditis and pericarditis by Gd-DTPA enhanced magnetic resonance imaging. Eur Heart J. 1994;15:283–284.
10. Scott DB, Lee A, Fagan D, Bowler GM, Bloomfield P, Lundh R. Acute toxicity of ropivacaine compared with that of bupivacaine. AnesthAnalg. 1989;69:563–569.
11. Coven G, Arpesella R, Ciceri M, Preseglio I, Cardani A. Accelerated idioventricular rhythm during spinal anesthesia for cesarean section. Int J ObstetAnesth. 2003;12:121–125.

12. Cotileas P, Myrianthefs P, Haralambakis A, Cotsopoulos P, Stamatopoulou C, Ladakis C. Bupivacaine-induced myocardial depression and pulmonary edema: a case report. J Electrocardiol. 2000;33:291–296.
13. Ryu HY, Kim JY, Lim HK, Yoon J, Yoo BS, Choe KH, Lee SH. Bupivacaine induced cardiac toxicity mimicking an acute non-ST segment elevation myocardial infarction. Yonsei Med J. 2007;48(2): 331-336.
14. Jin Yong Park, Hwa Rim Kang, Jee Hyun Kim et al. Fatal Peripartum Cardiomyopathy after Bupivacaine Local Injection in Elective Cesarean Section: A Case Report.Journal of The Korean Society of Emergency Medicine. 2016;27(2): 214-218.