

Original article:

Bupivacaine induced second degree atrioventricular block – a case report

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ABSTRACT:

Atrioventricular block (AV) is an emergency medical condition needing insertion of cardiac pacemakers temporally or permanently. The reversible causes of AV block include drug induced, autonomic disturbances, metabolic and endocrine causes. The drugs commonly responsible for AV blocks are beta blockers, calcium channel blockers, adenosine, lithium, digitalis and many of the anti-arrhythmic drugs. Bupivacaine a local anesthetic agent has been associated with cardiotoxicity. There are few case reports of cardiac conduction abnormalities associated with bupivacaine injection for local and spinal anesthesia. Here we present a patient with second degree AV block (Mobitz type II) associated with bupivacaine injection.

INTRODUCTION

Bupivacaine a white crystalline powder soluble in water is used for local and spinal anesthesia. It acts by blocking the generation and conduction of nerve impulses by increasing the threshold for electrical excitation and slowing the propagation of the nerve impulse, and by reducing the rate of rise of the action potential. Toxicity of bupivacaine is associated with cardiac and central nervous system manifestations. Cardiac manifestations include conduction abnormalities, hypotension and even sudden cardiac arrest. CNS effects may cause stimulation or depression. Most of the recent animal studies have

shown that effects occur after unintended intravenous absorption of bupivacaine

CASE REPORT:

A 26 year old female patient, G3P2L1D1 with 38 weeks of gestation was admitted for safe confinement. Patient was planned for elective lower segment cesarian section (LSCS) because of previous LSCS. Patient was not a diabetic, hypothyroid or with cardiac disease in past. On admission patient was conscious and oriented, no pallor, vitals were stable and systemic examination was normal. Complete haemogram, renal and liver function tests, serum electrolytes and thyroid profile were normal.

Patient was posted for LSCS with spinal anesthesia using inj. 2% lignocaine and 0.5% bupivacaine about 12ml at L3-L4 disc space and a male baby was delivered. Post procedure patient developed

bradycardia with a heart rate of 38 beats/ min about 3 hours after surgery. Electrocardiogram of the patient showed mobitz type II second degree AV block.

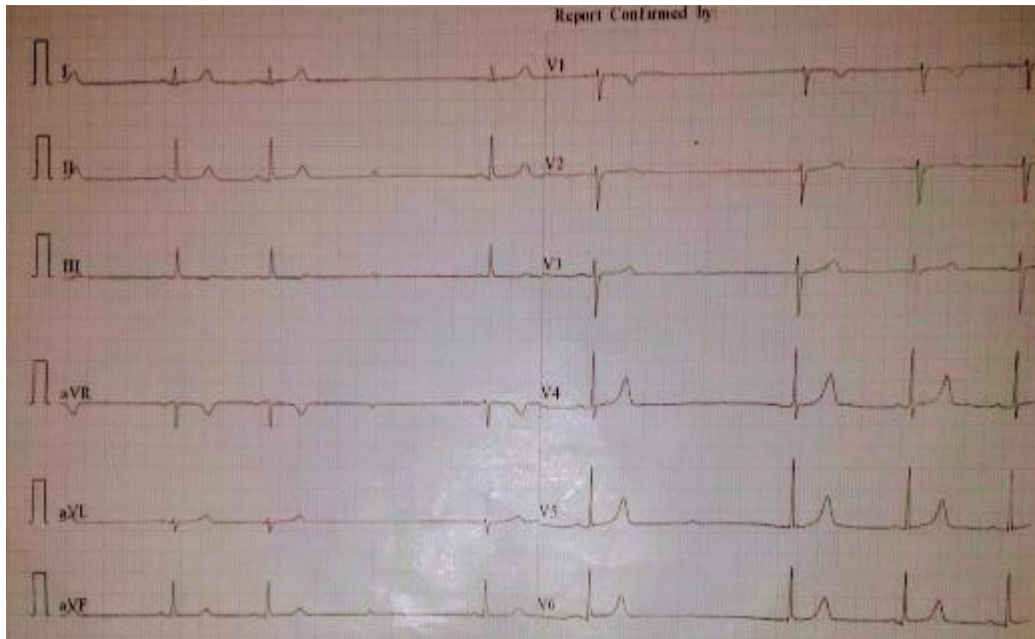


Image 1: ECG of the patient showing second degree AV block - Mobitz type II

Patient was asymptomatic inspite of AV block and the patient was monitored with continuous cardiac monitoring and after 3 hours patient again restored to normal sinus rhythm. When patient went into AV block the electrolytes and renal function tests were repeated and were normal. 2 Dimensional echocardiography was normal. After a week of hospital stay patient was discharged. The transient occurrence of the cardiac conduction abnormality here was attributed to the anesthetic agent bupivacaine.

DISCUSSION:

Atrioventricular(AV) block occurs due to disturbances in the conduction of electrical activity in and around the AV node. It is classified based on the

ECG findings, **First degree AV block** were PR interval prolongation is present but all P waves are conducted. In second degree heart block, there is progressive prolongation of the PR interval followed by a missed beat **Mobitz type I (wenckebach)**. In **Mobitz type II** there is intermittent conduction failure of the P wave without change in the preceding PR interval. There is complete failure of impulses from atrium to ventricle in **Third degree (complete) AV block..**

Artificial pacemaker implantation is the treatment for AV blocks, but all patients do not require pacemakers. Pacemaker implantation is required in symptomatic bradycardia (syncope, giddiness, heart failure) in all types of AV blocks,

patient on an essential drug causing bradycardia, period of asystole >3s or escape rate <40/min and neuromuscular diseases regardless of symptoms.

Bupivacaine hydrochloride is a local anesthetic related to mepivacaine and lidocaine. It acts by increasing the threshold for excitation, slowing conduction and reducing the rate of rise of action potential. It acts by blocking the inward current voltage-gated sodium channels. Its onset of action is rapid with peaking in blood by 30 to 45 minutes and duration of action is longer with lasting up to 7 hours. It is metabolized in the liver and excreted by the kidneys. Its pharmacokinetics is altered by renal and liver disease, route of administration, age of the patient and addition of epinephrine. Bupivacaine is contraindicated in patients with hypersensitivity and in paracervical block in obstetrics.

Adverse effects of bupivacaine involve the central nervous system (CNS) and cardiovascular system (CVS). These adverse effects from various studies have been transient to fatalities. The CNS adverse effects may result in CNS stimulation or depression. The cardiac toxicities include reduced myocardial contractility and conduction abnormalities. The adverse effects are usually seen after bupivacaine overdose in local anesthesia or accidental intravascular injection and addition of other local anesthetic or epinephrine. In case of spinal anesthesia the cardiac adverse effects manifest when the drug reaches T3-T4 spinal level. The possible mechanism is blocking of the sympathetic activity and vasovagal attack. Also the underlying cardiac status of the patient is important.

Coming to the cardiac conduction abnormalities there are case reports of arrhythmias and AV blocks in

patients due to bupivacaine toxicity. The arrhythmias reported in literature due to bupivacaine toxicity are ventricular tachycardia and accelerated idioventricular rhythm.

Animal model developed hypotension, ventricular tachycardia & fibrillation and respiratory arrest after bupivacaine administration. In human, 12 volunteers received i.v bupivacaine, and they developed depression of conductivity and contractility. Coven et al reported two cases with an accelerated idioventricular rhythm during spinal anesthesia using bupivacaine for caesarean section.

All three types of AV nodal block has been reported in literature and our patient with no previous comorbidities had asymptomatic second degree AV block (Mobitz II). In our case it was transient and patient restored to sinus rhythm spontaneously. But there are case reports where patient needed intervention. There is a case report by Hsuan Shih Chen and colleagues of bupivacaine induced junctional bradycardia successfully reverted by lipid emulsion. There are also case reports of myocardial depression associated with bupivacaine toxicity. There is a case report of myocardial depression presenting in 22 year old female post bupivacaine anesthesia presenting with ST elevation in ECG with reduced ejection fraction restored to normal within days with symptomatic management.

To conclude bupivacaine a commonly used local anesthetic agent can be associated with cardiac adverse effects and careful assessment of the patient cardiac status is necessary preanesthesia. Management of such cases depends on the patient symptomatology and hemodynamic status.

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