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Case Reports

SUDDEN VENTRICULAR FIBRILLATION DURING CATHETERIZATION DUE TO LIDOCAINE INFILTRATION FOR LOCAL ANESTHESIA IN A 53 YEAR OLD WOMAN

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ABSTRACT

Mrs. Heidari, 53 developed ventricular fibrillation and cardiopulmonary arrest after receiving 15 mL lidocaine 2% for local anesthesia at the Dr. Shariati Hospital Cathlab. There was no identified risk factor for her arrest preoperatively. She had a history of exertional chest pain for about 5 years and an electrocardiogram showed only inverted T-waves at the time of angiography. The case is reviewed in detail and a discussion of lidocaine toxicity is presented.

Keywords: Lidocaine, toxicity; Ventricular fibrillation

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INTRODUCTION

Lidocaine was introduced in 1948 and since then it has been the most widely used local anaesthetic around the world. This drug inhibits neural impulse generation by blocking sodium ion transport across the cell membranes by partial depolarization of the membrane.

Lidocaine has very few untoward effects on the cardiovascular system. The drug's major toxic effects are those of the CNS, including respiratory depression, delirium, agitation and convulsions. Although lidocaine overdosage may result in ventricular fibrillation and death, most of the catastrophic arrhythmias occurring after its use result from accompanying sympathomimetic effects.

Case Report

Mrs. Marzieh Heidari 53, was born and lived in Tehran. Since 1990, she complained of exertional chest pain radiating to her back and left arm which could be relieved by sitting. In December 1990, she was admitted to CCU for the first time becaused of unstable angina. Her electrocardiogram showed T-wave changes in precordial leads. The concurrent cardiac enzyme assays showed normal values. After her fifth day in CCU, she was discharged free of any complications.

In January 1991, she was admitted to CCU for the second time. This time the electrocardiogram showed inverted T-waves in leads V_1 to V_4. The cardiac enzyme levels on 3 successive days were as follows (she had had an intramuscular injection):

	day 1	day 2	day 3
CPK	14	262	21
LDH	99	213	104
SGOT	26	30	24

The patient was discharged after 5 days without any complications receiving nitrates, a beta-blocker and aspirin for drug therapy. One month later she was again hospitalized and coronary angiography was recommended. Physi-

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cal examination was normal and routine laboratory studies were within the normal range except for triglycerides: 388 and cholesterol: 215. ECG showed normal sinus rhythm, normal axis and inverted T-waves in leads V_1 to V_6.

At catheterization, after routine prep and drape the patient received a lidocaine injection for local anesthesia, following which she developed sudden cardiopulmonary arrest. CPR procedure was immediately initiated, and she was successfuly resuscitated and transferred to the ward. No immediate etiology as to her cardiopulmonary arrest was suspected. Angiography was not performed. After 4 days, she was discharged from the hospital on propranolol, nitrocontin and diazepam.

In September 1994, she was admitted to Shariati Hospital to undergo coronary angiography. Physical examination and routine laboratory tests were normal. ECG showed T-wave changes in precordial leads. Taking her previous arrest into account, the patient received 100 mg hydrocortisone and 25 mg promethazine before catheterization. After prep and drape 15 ml lidocaine 2% was injected subcutaneously in the right inguinal area, and femoral artery was punctured and a no. 8 arterial sheath introduced without any pain or distress. Meanwhile the vital signs of the patient were carefully monitored. At this time she suddenly developed ventricular fibrillation and cardiopulmonary arrest. After 200 J DC shock, the rhythm converted to normal sinus and the patient regained consciousness. Her blood pressure was 130/70 mmHg.

Selective coronary angiography and LV injection was performed with pigtal and right and left Judkins catheters and contrast material (Omnipaque). Results were a normal ejection fraction and 40% stenosis after the second septal branch of the left anterior descending coronary artery. After catheterization, the patient was transferred to the ward without any further complications and was discharged the next day.

DISCUSSION

Pharmacology

Lidocaine was originally introduced as a local anesthetic and antiarrhythmic agent in the early 1960s. Its primary use as an antiarrhythmic agent is in treatment of ventricular arrhythmias after myocardial infarction and cardiac surgery. Its local anesthetic action is mediated through membrane Na^+ channel blockade, following which propagation of nerve signals across the blocked area is no longer possible. Sodium channels which are in the resting state have a lower affinity for lidocaine blockade, therefore rapidly firing nerves are affected first.[1]

Interestingly, the cardiac antiarrhythmic effects of lidocaine are also mediated through Na^+ channel blockade. In fact, this appears to be the sole pharmacological effect of the drug. Lidocaine is classified as a class IB antiarrhythmic agent and is capable of blocking Na^+ channels in both resting and active states, while quinidine acts on channels which are in the active state.[4]

Untoward effects

Lidocaine's side effects are primarily those of the CNS. The major symptoms are a feeling of dissociation, perioral paresthesia, nausea, light-headedness, tremor, speech disturbances, drowsiness, hearing disturbances, agitation and convulsion.

Currently, lidocaine is considered the least cardiotoxic antiarrhythmic drug. There have been reports of exacerbated cardiac arrhythmias in less than 10% of patients. It is also considered to have a negative inotropic effect on the myocardium which is said to be due to decreased efficiency of oxygen utilization by the myocardium.[3]

There are few reports from across the world about inexplicable sudden cardiac arrest after infiltration of lidocaine, but no report of ventricular fibrillation has been made. In a report by Mishima in 1989, a 65 year old woman experienced cardiac standstill following an infiltration of lidocaine for local anesthesia. Electrocardiographic and laboratory studies revealed no predisposing abnormality.[8]

Although these side effects are not frequent enough to affect the common clinical usage of lidocaine as both an anesthetic and antiarrhythmic drug, these reports are still worth being considered more seriously. Not all instances of such cardiac arrests may occur in such clinical situations that a successful CPR could be performed. Also these case reports do not warrant having a CPR team available for every instance of lidocaine administration as a local anesthetic, but it may be justifiable to have such cases fully examined to find out any other possible factors that may have been responsible for the cardiac arrest; and if the cardiac arrest is attributable solely to lidocaine, what are the possible mechanisms, and more importantly, who are at risk?

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