

SINUS ARREST DUE TO LIDOCAINE

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SYNOPSIS

Sinus arrest culminating in grand mal fits, respiratory arrest and death resulted after 50 mg of Lidocaine was given intravenously to a patient with anterior myocardial infarction. This is a rare but serious complication of Lidocaine therapy and is potentially fatal as is illustrated in this case.

INTRODUCTION:

Sinus arrest following the administration of intravenous Lidocaine is a rare but serious complication which is potentially fatal. In this paper we report a patient who succumbed to this complication.

CASE REPORT

A 52 year old Chinese man was admitted to hospital because of severe chest pain. The 12 lead electrocardiogram showed recent transmural anteroseptal infarction with complete right bundle branch block. The frontal plane axis was around + 100 degrees indicating no coexisting hemiblock.

Two hours after admission to hospital, he was found to be breathless and clinical examination revealed signs of mild left heart failure. The blood pressure at that time was 142/86 mmHg. Because of a tachycardia, an electrocardiogram was done. Figure 1 (upper panel) shows a long strip of lead V1 which was recorded. This was interpreted by the house physician on call as showing ventricular tachycardia and 50 mg of Lidocaine was given intravenously over a one minute period. Two minutes after the termination of the injection, the patient complained of severe dizziness. The electrocardiogram (lower panel) then showed a severe bradyarrhythmia with hardly any discernable P waves. The ventricular rate was approximately 25/min and the ventricular complexes were widened. Soon afterwards, the patient developed grand mal convulsions and respiratory arrest and died despite attempts at cardio-pulmonary resuscitation.

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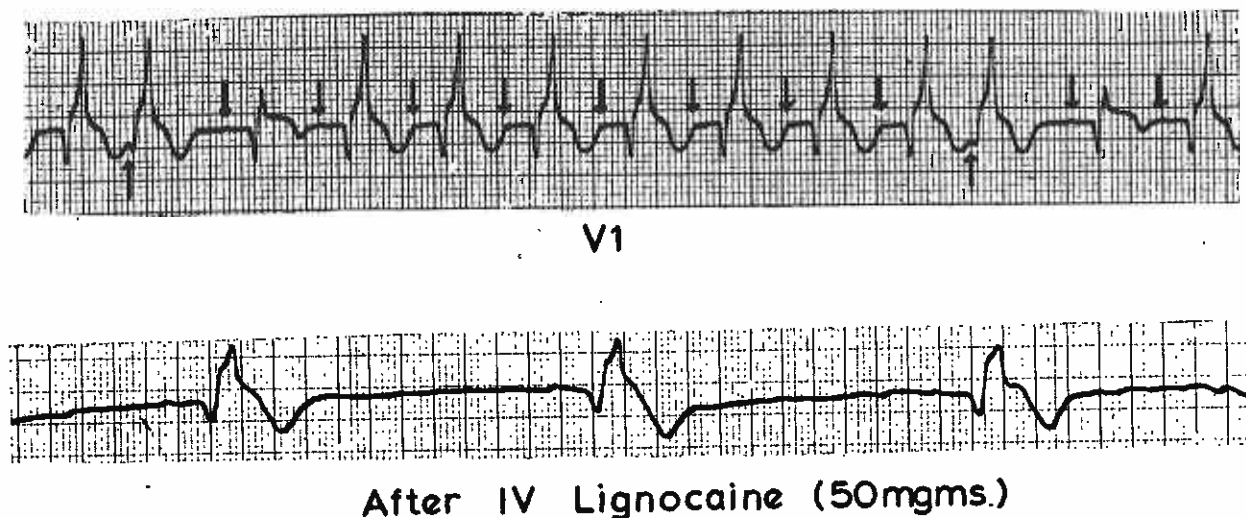


Figure 1 Top panel. Electrocardiogram (lead VI) shows anteroseptal infarction with tachycardia dependent right bundle branch block. The sinus rate is approximately 100/min (downward pointing arrows indicate sinus P waves). Two junctional ectopic beats are seen (Upward pointing arrows). After these ectopic beats, there is a pause and the sinus beats which follow show QRS complexes which are not widened. Bottom panel. After 50 mg of intravenous Lignocaine, there is sinus arrest, with a ventricular rate of approximately 25/min (electrocardiogram recorded at lead V1).

DISCUSSION

Lidocaine is the most commonly used antiarrhythmic agent for the treatment of ventricular arrhythmias. It is today widely accepted as a safe and effective antiarrhythmic drug (1). Recently, it has been suggested by several workers that Lidocaine should be given routinely as a prophylaxis against ventricular fibrillation during the acute phase of myocardial infarction (2). However, like all pharmacological agents, Lidocaine is not without dangerous side effects. One of these is the occurrence of sinus bradycardia or sinus arrest following its administration. So far, several cases of this serious complication have been reported in the literature. According to Jeresaty and co workers, (3) sinus arrest following Lidocaine has occurred, in general, under one of the following three conditions (A) as a probable interaction of Lidocaine with some other antiarrhythmic agent. For example, in the case reported by them, the patient was receiving Quinidine before Lidocaine was given. In the patient described by Wood, (4) sinus arrest followed administration of intravenous Phenytoin at a time when the patient was already receiving an intravenous infusion of Lidocaine. (B) in the sick sinus syndrome. In the patient described by Lippestad and Forfang, (5) sinus nodal dysfunction was apparent from the electrocardiographic recordings before Lidocaine was given (C) in inferior infarction. In this situation, sinus bradycardia is frequently already present, and it is therefore logical to assume that sinus arrest may occur more easily. In the case reported by Wood, (4) the patient had suffered a recent inferior infarction.

However, three cases of sinus arrest following the administration of Lidocaine alone have also been reported (6, 7). In none of these was there apparent sinus nodal dysfunction. One patient had acute myocardial infarction (site not stated) (6), another old inferior infarction (7), and the last coronary artery

disease (6).

The patient reported in this paper is interesting for several reasons. First, he was suffering from anterior and not inferior myocardial infarction. Second, no other drugs was given before Lidocaine was administered. Third, he appeared to have a normally functioning sinus node because the initial sinus rate was approximately 100/min. Reexamination of the electrocardiogram revealed that the right bundle branch block was tachycardia dependent, because following a junctional ectopic beat (second and eleventh beats in the upper panel) and a compensatory pause, the following sinus beat showed the pattern of transmural infarction, but the ventricular complex was not widened. Lastly, sinus arrest followed a conservative dose of Lidocaine (50 mg) given slowly over one minute.

In all the cases reported so far (1-6), the bradyarrhythmia improved after Lidocaine was stopped. In our patient however, respiratory arrest and grand mal fits unfortunately developed soon after the sinus arrest, and the patient succumbed despite cardiopulmonary resuscitation.

This report should in no way dampen the enthusiasm for the use of Lidocaine which is an extremely valuable drug for the treatment of ventricular arrhythmias. The purpose of documenting this patient is to alert others to a rare but potentially fatal reaction to one of the most commonly used drugs in cardiovascular therapy.

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