LIDOCAINE-INDUCED CARDIAC ASYSTOLE

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Intravenous administration of a single 50-mg bolus of lidocaine in a 67-year-old man resulted in profound depression of the activity of the sinoatrial and atrioventricular nodal pacemakers. The patient had no apparent associated conditions which might have predisposed him to the development of bradycardias; and, thus, this probably represented a true idiosyncrasy to lidocaine.

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References


The safety of intravenous therapy with lidocaine is a major reason for its popularity. Toxic effects are related to dosage and are most commonly due to stimulation of the central nervous system, although hemodynamic impairment has also been reported with very high levels in the blood.1-3 We report herein a rare complication of therapy with lidocaine, ie, profound depression of the automaticity of the sinoatrial and atrioventricular nodes.

Case Report

A 67-year-old man, who had previously been healthy and was taking no medications, was admitted to the hospital with a 24-hour history of episodes of dizziness and light-headedness, each lasting less than one minute. The findings from physical examination were normal, except for an irregular pulse rate of 80 beats per minute. The blood pressure was 130/90 mm Hg, and the patient’s weight was 79 kg (174 lb). An electrocardiogram showed frequent ventricular premature contractions but was otherwise normal. The hemoglobin level, the white blood cell count, the erythrocyte sedimentation rate, the serum levels of sodium, potassium, chloride, and creatinine, the blood gas levels, and the chest x-ray film were within normal limits.

Three hours later, because of the increasing frequency of ventricular premature contractions, 50 mg of lidocaine was administered intravenously over 90 seconds. Within 30 seconds the patient developed sinus bradycardia, followed by sinus standstill and junctional escape at a rate of 32 impulses per minute; and this, in turn, was followed by depression of the junctional rhythm, with episodes of asystole lasting several seconds (Fig 1). Atropine sulfate (0.4 mg) was administered intravenously, and over the next five minutes, there was a gradual return to sinus rhythm at a rate of 80 beats per minute.
This was the only episode of bradycardia seen during five days of electrocardiographic monitoring, despite the fact that, because of persisting ventricular premature beats, quinidine and then procainamide were administered orally after a prophylactic temporary transvenous pacing electrode had been placed in the right ventricle. No enzyme evidence of myocardial infarction was detected, and the ECG remained normal. The patient was discharged after ten days of therapy with procainamide (500 mg orally four times daily). Three months later, he remained asymptomatic while receiving the same drug.

**DISCUSSION**

At therapeutic levels in the blood, lidocaine has no effect on the automaticity of the sinoatrial and atrioventricular nodes, the time for sinoatrial recovery, and atrial and atrioventricular nodal conduction time and refractoriness; however, at concentrations in the plasma above 5 μg/ml, bradycardia does occur in both animals and man. The electrophysiologic basis for this has been studied in atrial preparations from rabbits by Mandel and Bigger, who observed both depression of the automaticity of sinoatrial nodal cells and sinoatrial exit block, occurring only at concentrations of lidocaine considerably in excess of those required to cause significant effects on ventricular muscle and Purkinje fibers. In patients with the "sick sinus syndrome," Ross and Dunning found that therapeutic doses of lidocaine had no effect on sinoatrial or atrioventricular nodal function.

Lidocaine has been reported to have caused profound bradycardia in only six patients previously. Of these, three were receiving other drugs with depressive effects on sinus nodal function, and two had sinus nodal depression as part of their disease, and two may have developed high levels of lidocaine in the blood due to the
presence of congestive heart failure in both patients and the use of large doses of lidocaine in one.1,2
It is inconceivable that the single 50-mg bolus of lidocaine that our patient received could result in a toxic level in the blood. His sinoatrial nodal function was not tested formally, but the absence of recurrence of the sinus bradycardia, despite the administration of quinidine and procainamide (drugs which usually depress sinoatrial nodal function much more than lidocaine), suggest that there was no underlying abnormality of the sinoatrial node. Thus, the reaction to therapy with lidocaine likely represents a true idiosyncrasy.

The role of atropine in the treatment of this patient’s bradycardia is uncertain, since lidocaine is not known to affect parasympathetic activity. Although recovery occurred within minutes of the injection of atropine, this might be explained by the rapid fall in the level of lidocaine in the blood due to distribution out of the intravascular compartment.3,4

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REFERENCES

PNEUMOMEDIASTINUM

An Unusual Complication of Needle Biopsy of the Lung

David L. Klein, M.D., and Gordon Gameu, M.D.

PNEUMOMEDIASTINUM without pneumothorax is an unusual and apparently benign complication of needle biopsy of the lung.

Biopsy of the lung performed by means of percutaneous aspiration via a needle1,2 has become increasingly accepted as a diagnostically reliable and generally safe procedure for investigating selected intrathoracic lesions. Common complications are pneumothorax, local pulmonary bleeding, and hemothysis. We encountered the unusual and apparently benign complication of pneumomediastinum resulting from needle biopsy of the lung.

CASE REPORT

A 54-year-old woman with squamous cell carcinoma of the tongue had a chest x-ray film that demonstrated a single, 2-cm cavitated nodule in the left upper lobe. Under fluoroscopic guidance, transthoracic biopsy was performed by inserting a 19-gauge spinal needle into the lesion and using a Nordenström stylet to disrupt cells from the lesion. Cytologic examination of the specimens obtained disclosed squamous cell carcinoma.

The patient tolerated the biopsy without difficulty. A chest x-ray film obtained immediately after biopsy showed no pneumothorax or pneumomediastinum. A chest x-ray film taken four hours later demonstrated pneumomediastinum with emphysema of the soft tissue but no pneumothorax (Fig 1). The pneumomediastinum and subcutaneous gas resolved spontaneously and without treatment.

DISCUSSION

Sinner3 described the complications occurring in 5,300 percutaneous transthoracic biopsies via needle aspiration in 2,726 patients over a 14-year period. The most common complication was pneumothorax; it occurred in 744 patients, and only 8 percent of these patients required treatment. Pneumomediastinum alone occurred in only two of the 2,726 patients, and neither patient required treatment.

There are several possible explanations for the pneumomediastinum in our patient. First, development of

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