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Treatment of Perinfarction Recurrent Ventricular Fibrillation by Percutaneous Pharmacological Block of Left Stellate Ganglion

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Summary: A patient suffering from an acute myocardial infarction presented on the seventh and eighth days of hospitalization recurrent episodes of ventricular fibrillation refractory to antiarrhythmic treatment. The life-threatening ventricular fibrillation was suppressed by percutaneous pharmacological block of the left stellate ganglion.

Key words: acute myocardial infarction, ventricular fibrillation, left stellate ganglion

Introduction

It has been postulated than an imbalance of the sympathetic activity may play a major role in the genesis of malignant arrhythmias occurring in the early phase of acute myocardial infarction (Schwartz *et al.*, 1975). The present report describes the clinical history of a patient suffering a large acute myocardial infarction who presented on the seventh and eighth days with recurrent ventricular fibrillation, refractory to antiarrhythmic treatment, which was eventually controlled by the pharmacological block of the left stellate ganglion.

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Case Report

A 56-year old-man, with a history of effort angina, suddenly experienced chest pain radiating to the neck and to the left arm, partly sensitive to sublingual administration of nitroglycerin. He was admitted to our CCU about 20 hours after the onset of symptoms. On admission chest pain was still present and required administration of morphine. Physical examination revealed tachycardia and mild rales in the lower lung fields. Electrocardiogram showed ST-segment elevation in leads V_{2-5}, pathological Q waves in leads V_{1-3}, long QT interval (0.48 s according to Barett's formula), and normal PR interval (0.16 s). At echocardiogram asynergy of the septum and anterior wall without left ventricle enlargement was present. The first serum enzyme activity determination showed a creatine phosphokinase value of 3062 IU/l, which was the maximum value reached during hospitalization. No evident alterations were apparent on the standard chest x-ray. Electrocardiographic monitoring revealed the presence of isolated premature ventricular contractions (PVC) which were suppressed by infusion of lidocaine 2 mg/min (the infusion was stopped on the fourth day). Isosorbide dinitrate 5 mg/h was also started on admission and was continued for 11 days with a dosage ranging between 4 and 8 mg/min according to the symptoms experienced by the patient.

On the sixth day after admission the clinical condition of the patient was stable: at ECG pathological Q waves in leads V_{1-5} were present, the ST segment was still elevated in the same leads but less so than at admission, the QT_c interval was still long (0.48 s), and the PR interval was normal (0.16 s). Repeated echocardiograms had shown an extension of asynergy to the apical portion of

the lateral wall. The cavity of the left ventricle was slightly greater than at admission and the presence of mural thrombi was obvious. Chest x-ray film was normal and creatine phosphokinase activity was 170 IU/l, the level of the serum electrolytes was in the normal range. The patient was still on nitrate infusion in spite of the fact that he experienced the last chest pain 3 days before. During the night, while sleeping, the patient presented runs of PVCs, both repetitive and premature, and thereafter ventricular fibrillation. Prior to the arrhythmias the patient did not experience chest pain and any apparent ischemic changes were present on the electrocardiogram. Sinus rhythm was restored after three direct current shocks combined with bolus administration of 100 mg lidocaine. Lidocaine infusion (2 mg/min) was started, but a second episode of ventricular fibrillation occurred 1 hour later. Direct current cardioversion was required to restore the sinus rhythm. In the 12 hours following lidocaine and nitrate infusion, 2 more episodes of ventricular fibrillation occurred, all were successfully treated by electrical cardioversion. Therefore, an infusion of 17-mono chloride acetyl ajmaline (Lorajminum, Ritmos Elle®) (0.7 mg/min), preceded by a bolus of 100 mg, was started. Nevertheless, in the following hour the patient experienced four more episodes of ventricular fibrillation, all requiring electrical cardioversion.

Because of the apparent inefficacy of the drug administration and considering the marked left ventricular dysfunction which advised against the use of antiarrhythmic agents such as procainamide or quinidine with a more pronounced negative inotropic effect, we considered the possibility of attempting a pharmacological block of the left stellate ganglion by local anesthetic. This was performed by percutaneous administration of 120 mg of lidocaine. After the administration of the anesthetic a ptosis of the left eyelid was present and an immediate and complete disappearance of PVCs occurred.

The patient, still under antiarrhythmic infusion at the same rate, did not experience any other episode of ventricular fibrillation until 11¼ and 11½ h after ganglion block. At these times the entity of the pharmacological block of the ganglion activity was probably reduced as judged by the disappearance of the ocular signs, and antiarrhythmic therapy was ineffective in preventing the episodes of ventricular fibrillation. The pharmacological block was therefore repeated with lidocaine, and 5 hours later mepivacaine, a more long-acting anesthetic, was administered. Mepivacaine was given again after 18 and 30 h (Fig. 1). In this period the ocular signs of ganglionic block were constant and only rare PVCs were observed. The electrocardiograms obtained some hours after the pharmacological ganglion block, but not at scheduled intervals, did not show any appreciable change in the QT_c interval or in the PR interval (the QT_c being between 0.50 and 0.48 s and the PR 0.16 s).

A few hours after the first local application of mepivacaine the patient was withdrawn from the antiarrhythmic drug but the infusion of nitrates was continued. He experienced no more arrhythmias. After discharge the patient was followed for 6 months during which time he underwent two periods of 24-h Holter monitoring that revealed the absence of any arrhythmias.

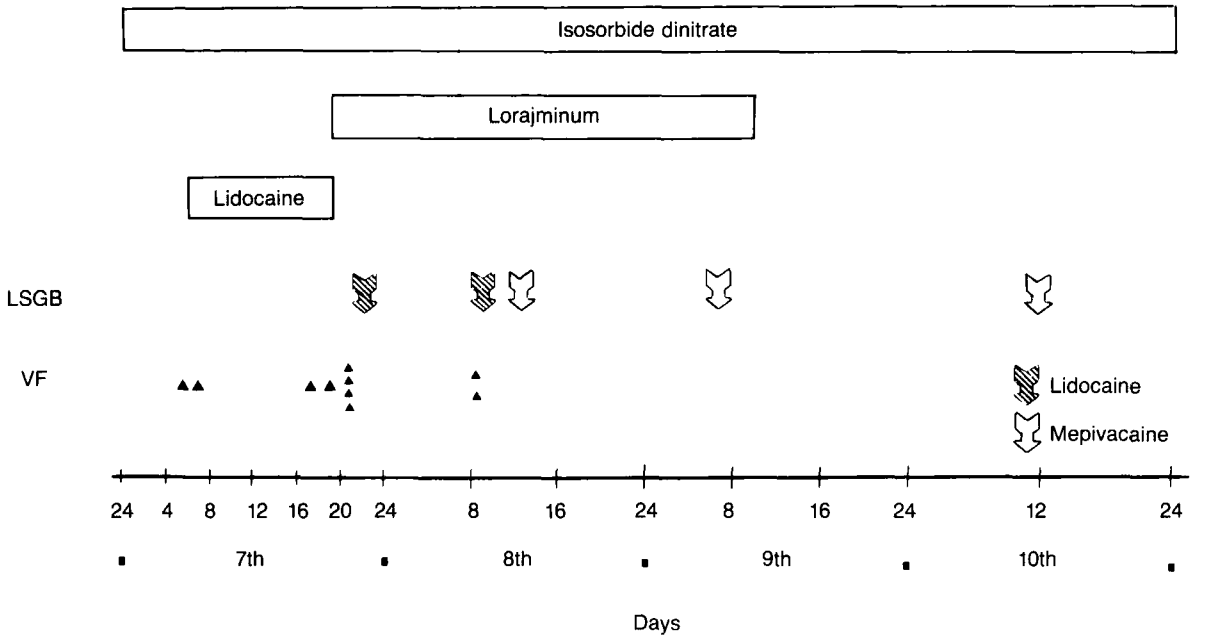

FIG. 1 The pharmacological treatment and the percutaneous administration of a local anesthetic in order to obtain the left stellate ganglion block (LSGB) on the days in which the patient experienced the episodes of ventricular fibrillation (VF) are presented in this figure.

Discussion

Animal models have demonstrated that an imbalance in the sympathetic drive to the heart may play a major role in the genesis of life-threatening arrhythmias (Schwartz and Stone, 1980; Schwartz *et al.*, 1975). That this phenomenon can also be present in humans has still to be demonstrated with the exception of patients with long QT syndrome (Schwartz *et al.*, 1976). The object of our report is a patient who after an acute anterior myocardial infarction showed long QT_c interval and presented several episodes of ventricular fibrillation on the seventh and eighth days of hospitalization despite antiarrhythmic drug administration. The pharmacological block of the left stellate ganglion via percutaneous application of lidocaine was temporarily able to interrupt the occurrence of ventricular fibrillation. This occurred again after almost 12 hours when the level of the sympathetic block probably became insufficient, as indicated by the disappearance of the ocular signs during the infusion of antiarrhythmic drug.

This suspicion was paramount in our decision to use mepivacaine rather than lidocaine, which has a longer duration of action, to anesthetize the left stellate ganglion. Apparently this was a proper choice, as ventricular fibrillation did not occur during and for 2 days after the block.

In addition, we cannot exclude a direct antiarrhythmic effect of the anesthetic injected subcutaneously, because the serum level of the drug was not measured. We believe this hypothesis is very weak, considering that the intravenous administration of the drug was not able to prevent the episodes of ventricular fibrillation. The apparent time relationship between the occurrence of ventricular fibrillation and degree of left stellate ganglion block, as assessed by the presence of ocular markers, suggests that in humans in the acute phase of AMI an imbalance in sympathetic drive to the heart may be responsible for life-threatening arrhythmias. Indeed, we were not able to observe a significant QT interval shortening after the left stellate ganglion pharmacological block, but this is probably due to the fact that the electrocardiograms were obtained some hours after the procedure and not at scheduled time intervals.

In conclusion, considering the low risk, in experienced hands, of percutaneous pharmacological block of the left stellate ganglion, it might be worthwhile in patients with life-threatening arrhythmias not controlled by antiarrhythmic drugs to attempt the pharmacological block of the activity of the left stellate ganglion. As in the case presented in this report, it may be possible to overcome a very critical clinical condition without being forced to use antiarrhythmic drugs with a possible harmful negative inotropic effects.

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