

The Use Of Intravenous Streptokinase In A Rural Community Hospital

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Abstract: To determine the safety and efficacy of the use of intravenous streptokinase in a 110-bed rural community hospital, we studied 28 consecutive patients with clinical and ECG evidence of acute myocardial infarction. Twenty (74 percent) of the 27 patients who had cardiac catheterization after treatment with intravenous streptokinase were found to have a patent artery supplying the infarcted area. One patient (3.5 percent) died of intractable heart failure, 2 (7 percent) had ventricular fibrillation, and 9 (32

percent) had ventricular tachycardia. Six patients (21 percent) had minor bleeding problems, and 1 developed a pseudoaneurysm at the catheterization site. We maintained close communication with a consulting cardiology group who provided additional medical or surgical therapy in a tertiary medical center for patients who needed it. We believe that intravenous streptokinase can be safely and effectively used in a rural community hospital. (JABFP 1988; 1:87-90.)

The use of intracoronary streptokinase (ICSK) to preserve cardiac function by allowing reperfusion of the myocardium during acute coronary occlusion has been the subject of many reports.¹⁻¹⁰ Intracoronary use necessitates the availability of a 24-hour cardiac catheterization laboratory and an experienced cardiologist. Many primary care hospitals, however, are without catheterization facilities, and transportation of the patient to a tertiary care center delays the administration of streptokinase and decreases the chances for myocardial salvage.¹ Moreover, transportation during an acute myocardial infarction may increase morbidity and mortality.¹¹

Intravenous streptokinase (IVSK) could be suitable for patients in hospitals having intensive care units (ICU) but no cardiac catheterization laboratories,¹¹⁻¹⁶ when staffed by physicians skilled in management of myocardial infarctions. Administration of IVSK requires continuous electrocardiograph (ECG) monitoring and routine stat lab services, but most important is the presence of interested physicians familiar with streptokinase complications who can communicate closely with referral cardiologists. After stabilization in a local ICU, patients whose ischemic muscle is reperfused by successful thrombolysis can be transferred with less risk and emotional turmoil to a referral center for further evaluation and therapy. This study re-

ports such experiences with IVSK in a rural, general hospital staffed mainly by family practitioners.

Methods

The study group consisted of 28 patients, aged 35-73 years, with clinical and ECG evidence of acute transmural myocardial infarctions, who came to the emergency room of our 110-bed, rural, general hospital. All patients with continuous chest pain unresponsive to sublingual nitroglycerin, and less than 6 hours in duration, were considered for IVSK therapy if their ECG met the appropriate criteria. Intravenous nitroglycerin was not used in this study. ECG criteria were S-T segment elevation of 0.2 millivolts or more in at least two precordial leads for anterior myocardial infarction, and both S-T segment elevation in inferior leads and reciprocal depression in anterior or lateral leads for inferior infarction. Patients with subendocardial infarction were excluded, as well as those with a history of recent gastrointestinal bleeding, stroke, surgery, or malignant hypertension. Our protocol (obtained from our consulting cardiology group) included a serum creatine kinase on admission and every 6 hours for the first 24 hours after streptokinase (SK) therapy, as well as daily ECGs for 3 days. Treatment for complications of acute myocardial infarction was not altered by thrombolytic therapy. Informed consent outlining possible complications and alternative therapy was obtained prior to infusion of SK. No patients during this time refused to participate.

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The drug was administered in the emergency room of our local hospital. A test dose of approximately 1,000 IU intravenous streptokinase was given over 5 minutes followed by a bolus of 500,000 IU and continuous infusion of streptokinase at 200,000 IU per hour for 4 hours. Then, a bolus of 5,000 units of heparin was given, followed by 1,000 units of heparin per hour by continuous infusion. Prophylactic intravenous lidocaine was also administered. All patients remained in the coronary care unit and were transferred by ambulance, with an ICU nurse in attendance, less than 24 hours after initiation of SK, to our tertiary care center about 50 miles away, where cardiac catheterizations were performed.

Results

Twenty-eight patients were given IVSK within 0.9 to 3.5 hours (mean = 2.8 hours, ± 1.13) from the onset of chest pain. Fifteen patients had ECG evidence of anterior wall myocardial infarction (MI), and 13 had evidence of an inferior wall MI.

A single death occurred, a 61-year-old woman with prior congestive heart failure who had a true posterior myocardial infarction. She presented in a cardiorespiratory arrest, and after successful resuscitation, she remained in cardiogenic shock. Because of her precarious status, she was not transferred to the tertiary hospital. IVSK was given in addition to pressor agents. She extubated herself on the second hospital day, suffered another cardiorespiratory arrest, and we were unable to resuscitate her again.

Peak creatine kinase in the survivors ranged from 259 to 6,000 (mean = 2,466, $\pm 1,378$). Seventeen patients had a class I Killip classification (absence of rales and S3), 5 patients had class II (presence of S3 or rales over 50 percent or less of lung fields), 3 patients had class III (rales over more than 50 percent of lung fields), and 3 patients presented in Killip class IV (shock).

Cardiac Catheterization Results

All surviving patients had cardiac catheterization. Of the 13 patients with an inferior wall myocardial infarction, 9 were found to have the right coronary artery patent, and 2 had the circumflex artery patent at the time of catheterization (two inferior myocardial infarctions were apparently caused by an occlusion of a dominant circumflex

system). Of the 15 patients with evidence of anterior wall myocardial infarction, 11 were found to have the artery supplying the area of previous infarction patent at the time of initial catheterization. The average time from onset of pain to the administration of streptokinase in the 5 unsuccessful patients was 3.3 hours, ± 1.28 hours, while the average time for the 22 successful patients was 2.7 hours, ± 1.13 hours. Lesions obstructing more than 50 percent of the coronary artery were present in 19 patients, and more than 70 percent obstruction was present in 17 of these patients. Nine patients had normal coronaries other than the previously occluded vessel. Ejection fractions obtained from catheterization ranged from 26 percent to 80 percent. At the time of catheterization, the area supplied by the previously occluded vessel appeared viable in 13 patients and was questionable in 10 patients and did not appear viable in 4 patients.

The mean length of time from the administration of IVSK to cardiac catheterization was 28.1 hours (range = 2–58.1 hours). For those with successful openings of the infarct artery, the mean was 27.4 hours; for patients with unsuccessful openings, the mean was 28.4 hours.

Complications

Six patients had bleeding complications, and 2 showed gross hematuria that cleared in 3 days or less. Three had hematomas of venipuncture sites; 1 had bleeding at the site of subclavian temporary pacemaker insertion with chest ecchymosis in addition to a pseudoaneurysm at the catheterization site, which required elective repair 6 weeks later. No patients required transfusions, and no cardiac hemorrhagic complications were noted.¹⁷⁻¹⁸

Two patients had ventricular fibrillation after streptokinase. Nine had ventricular tachycardia, and 1 had ventricular tachycardia both before and after streptokinase therapy. These arrhythmias were treated in the usual fashion with drug therapy and countershock when indicated.

Follow-Up

All surviving 27 patients have been followed between 9 and 44 months. One has had a recurrent myocardial infarction, which was treated again with streptokinase with good results. Twelve

patients had coronary artery bypass grafting after catheterization,¹⁹⁻²⁰ 3 had percutaneous transluminal coronary angioplasty, and 2 had both. Ten were treated medically. One patient is short of breath with minimal activity, 1 with moderate activity, and 2 with extreme activity. The others have no shortness of breath. Ejection fractions on the multiple-gated acquisition scan range from 20 percent to 79 percent (mean = 53 percent).

Discussion

In 28 patients who came to our hospital with acute myocardial infarction, 22 had patency of the infarct artery after thrombolysis. Five patients had occlusion of the infarct artery, and 1 patient died before cardiac catheterization could be performed. The patient died of intractable heart failure and arrhythmia and not as a result of the streptokinase therapy. Minor bleeding problems were encountered; the most serious complication was that of a pseudoaneurysm at the catheterization site, which required surgical repair 6 weeks later.

One might ask whether the infarct arteries were, in fact, occluded, since angiography was not done before IVSK. Our answer is that all patients had clinical evidence of acute myocardial infarction, and in other patients presenting with similar criteria, 9 percent were found to have an occluded artery.¹¹ In addition, the creatine kinase was elevated in all our patients. Moreover, we believe that the time from streptokinase administration to catheterization was short enough (mean = 28.1 hours) that spontaneous opening of the infarct artery was unlikely to have occurred in all our successful patients.

For those patients receiving IVSK, indirect non-invasive markers of coronary thrombolysis were used.²¹ These markers included reperfusion arrhythmias,²² rapid chest pain relief, early creatine kinase peaking,²³⁻²⁴ and rapid EKG changes to normal.²⁵ We could predict artery patency by using these data in most cases.

Successful preservation of myocardium following infusion of streptokinase during an acute myocardial infarction has been demonstrated and discussed in studies from tertiary centers where a catheterization laboratory and cardiologists are available. The purpose of this article is to demonstrate that successful results may also be obtained in a community hospital. Open

communication with a consulting cardiologist and a tertiary care center with catheterization facilities is necessary. Strict arrhythmia precautions must be maintained, and patients must be closely watched for early signs of excessive bleeding. In addition, diagnosis of an acute myocardial infarction must be secure, and no contraindications should exist. The family physician undertaking such treatment should be familiar with the literature about reperfusion during an acute myocardial infarction. Although the use of IVSK may seem simple, its use must never be casual. Early thrombolysis can result in greater reduction of myocardial infarct size and can be safely performed in rural community hospitals.

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RECERTIFICATION: SIX OR SEVEN YEARS?

All Diplomates are notified by routine mailing from the Board before the *sixth* year of certification that their certificates will be expiring at the end of the seventh year. Most Diplomates respond by taking the certification in their sixth year and a few wait until the seventh year to go through the recertification process. It is important that all Diplomates have the choice of taking recertification either in the sixth or seventh year; it makes no difference to the Board. However, reasons for not waiting until the seventh year are as follows:

If a Diplomate does not choose (or for any reason is unable) to sit for the recertification examination in the sixth year, or if a Diplomate sits for examination in the sixth year, but fails to make a passing score, then that Diplomate may reapply to participate in the recertification procedure the next (seventh) year. Thus, the Diplomate remains in good standing as a Diplomate at least for another year.

Should a Diplomate wait until the seventh year and not be recertified, for whatever reasons, then that Diplomate's certificate will expire December 31st of the (seventh) year noted on the current certificate, and the physician loses Diplomate status with this Board. The physician may reapply for the recertification process but must meet all the criteria that are applicable at the time of the reapplication.

A few Diplomates feel that if they take the recertification examination in the sixth year they are being "cheated" out of one year, to which the Board can only say that if such a feeling exists, then perhaps that Diplomate should wait until the seventh year. However, the certificate will be dated for a seven-year period from the year of recertification, no more, no less. We reiterate that the choice is strictly the Diplomate's and if one wishes to wait until the seventh year, that is entirely valid. The sixth-year option merely ensures that the Diplomate status remains intact for one year should the Diplomate miss or fail the examination.