ing IL-1 \textsuperscript{18,19} enters the thermoregulatory centers of the brain, infectious fever is believed to be due to the peripheral production of PGE\textsubscript{2}, which circulates centrally to act on the hypothalamus. \textsuperscript{18} We are unaware of any data suggesting a peripheral pyretic action of endogenous pyrogen; however, the fact that quadriplegics have febrile episodes at all in response to sepsis indicates that a peripheral pyretic action may be the source. Cimetidine has been described as causing fever, probably through a blockade of the H\textsubscript{2} receptors in the hypothalamus. \textsuperscript{20} Ranitidine, which this patient was receiving, may also have this effect.

This episode may represent a manifestation of NMS since hyperpyrexia and hemodynamic instability followed the administration of haloperidol by 12 h. Core features of NMS are pyrexia, altered consciousness, muscular rigidity, and autonomic dysfunction progressing over 24 to 72 h. \textsuperscript{18} The therapeutic action of neuroleptic drugs is believed to be through dopamine-receptor blockade in the basal ganglia and the hypothalamus; this central action is also believed by some to be the cause of NMS, a hypothesis that is supported by the occurrence in patients receiving dopamine-depleting drugs. \textsuperscript{8} A peripheral skeletal muscle mechanism similar to that seen with malignant hyperthermia has also been suggested, \textsuperscript{2} with support being provided by skeletal muscle pathology from victims demonstrating evidence of a toxic myopathy with absent muscular glycogen and lipid stores. \textsuperscript{16} In addition, dantrolene sodium, which acts peripherally on the contractile system of muscles, may be effective in some cases of NMS, \textsuperscript{10} and muscle contracture has been induced in vitro by another neuroleptic, chlorpromazine; \textsuperscript{7} however, muscle biopsy specimens from patients surviving episodes of NMS do not appear to give abnormal in vitro halothane-caffeine contracture tests. \textsuperscript{1}

The argument for a haloperidol alone causing the hypertensive episode is weakened by the record of a temperature of at least 41.2°C during the 24 h prior to the administration of haloperidol; because this episode was not accompanied by hemodynamic instability, it is likely that it was a continuation of his febrile course and not another hyperpyrexic event.

REFERENCES


Acute Myocardial Infarction Associated with Intravenous Injection of Pentazocine and Tripelennamine*

Bryan W. McGwier, M.D.; Martin A. Alpert, M.D., F.C.C.P.; Hercules Panagiotou, M.D.; and Charles R. Lambert, M.D., Ph.D.

This case report describes the evolution of an acute anteroseptal myocardial infarction in a 27-year-old man following intravenous injection of pentazocine and tripelennamine. Subsequent coronary angiography showed normal coronary arteries. Based on the known mechanism of action of these drugs, it is postulated that myocardial infarction resulted from coronary artery spasm secondary to excessive catecholamine stimulation.

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*From the Division of Cardiology, University of South Alabama College of Medicine, Mobile.

Reprint requests: Dr. Alpert, Division of Cardiology, 4th floor, Martin Bldg, Suite H, Mobile, Alabama 36617
I illicit drugs such as cocaine have been implicated as a cause of acute myocardial infarction. In this report, we describe a young man who developed acute myocardial infarction shortly after intravenous injection of pentazocine and tripelennamine.

CASE REPORT

A 27-year-old black man presented to the University of South Alabama Medical Center with a complaint of crushing substernal chest pain of 1-h duration. The chest pain started 15 to 30 min after injection of approximately 2 to 3 ml of a mixture of 150 mg of pentazocine and 50 mg of tripelennamine dissolved in tap water and filtered through cotton.

The patient was a known drug abuser, having previously used intravenous heroin and having injected pentazocine and tripelennamine intravenously on several occasions. He had no history of cardiovascular disease and had never developed chest pain following intravenous drug injection. His blood pressure on admission was 80/60 mm Hg, and his pulse rate was 110 beats per minute. The respiratory rate and body temperature were normal. Findings from the remainder of the physical examination were normal except for the presence of needle tracks on both forearms. The resting 12-lead electrocardiogram obtained on admission showed ST-segment depression in leads V1 to V6. The complete blood cell count, urinalysis, serum electrolyte levels, and chest x-ray film obtained on admission were normal.

This patient's blood pressure and heart rate rapidly normalized following intravenous infusion of 1 L of physiologic saline solution. Chest pain continued despite normalization of blood pressure and heart rate, but rapidly abated following intravenous infusion of nitroglycerin at a rate of 75 μg/kg/min. Beta-adrenergic blocking agents and morphine were not used. Following resolution of chest pain, 20 mg of nifedipine was administered orally every 6 h, and intravenous nitroglycerin was discontinued. There were no further episodes of chest pain during the hospitalization.

Follow-up electrocardiograms showed evolution of an acute Q-wave anteroapical myocardial infarction with associated lateral ischemia. The total serum creatine kinase level rose to a peak of 2,540 IU at 24 h, with an MB fraction of 14 percent. Toxicologic evaluation of whole blood tested positive for pentazocine and tripelennamine but negative for cocaine, opiates, and other commonly abused drugs. Diagnostic left heart catheterization and coronary angiography performed 7 days after the event showed mild anterolateral hypokinesia, a left ventricular ejection fraction of 50 percent, and a left ventricular end-diastolic pressure of 12 mm Hg. The coronary angiogram showed normal epicardial coronary arteries (Fig 1A and 1B). The patient's remaining course of hospitalization was uneventful.

DISCUSSION

In recent years, intravenous injection of pentazocine and tripelennamine has been used increasingly by drug abusers to achieve a heroin-like euphoria. This particular combination is frequently referred to as "Ts and Bs" or "Ts and blues," the "T" representing the first letter of the brand name, Talwin, and the "Bs" or "blues" referring to the color of tripelennamine tablets.

Intravenous injection of pentazocine and tripelennamine in combination has been reported to produce nausea, vomiting, headache, seizures, agitation, anxiety, muscle spasms, syncope and presyncope, and elevation of systolic and diastolic blood pressure. Chest pain has been previously described; however, this is the first reported case of acute myocardial infarction associated with intravenous injection of pentazocine and tripelennamine.

FIGURE 1. Coronary angiogram showing normal left coronary system and normal right coronary artery.

Pentazocine has both opioid agonist and weak antagonist properties. In patients with acute myocardial infarction, it has been shown to raise systemic vascular resistance, which may relate to its ability to increase serum catecholamine levels. Tripelennamine is a histamine receptor antagonist that also increases serum catecholamine levels. In addition, it exerts a cocaine-like effect by preventing reuptake of neurotransmitters in nerve endings.

Cocaine has been shown to block reuptake of norepinephrine in the synaptic clefts of a variety of tissues, including vascular smooth muscle and myocardium. Such blockade enhances adrenergic stimulation. In long-term cocaine abusers, depletion of dopamine prevents stimulation of dopamine-1 receptors, resulting in loss of the protective vasodilative effect. It is possible that pentazocine and tripelennamine might produce similar biochemical changes. Excessive adrenergic stimulation in affected individuals might lead to coronary artery spasm or excessive myocardial oxygen consumption (or both), potentially resulting in myocardial ischemia or infarction. If such is the case, then administration of β-adrenergic blocking agents would theoretically be...
contraindicated, due to resultant unopposed α-adrenergic stimulation. Administration of α-adrenergic blocking drugs (eg phentolamine), calcium-channel blocking agents, and possibly intravenous nitroglycerin or nitroprusside would be more rational therapeutic choices.

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Mitral and Tricuspid Annular Endocarditis*

Diagnosis by Transesophageal Echocardiography

Mohandas M., Shenoy, M.D.; and Kulandaielv Chandrasekaran, M.D.

Two cases of infective endocarditis with vegetations attached to the mitral and tricuspid annuli are described. In both cases, the vegetations could not be identified by transthoracic echocardiography. These cases illustrate the advantage of TEE over the transthoracic approach in recognizing vegetations in extravascular locations.

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TEE = transesophageal echocardiography

Infective endocarditis is a serious disease that requires prompt recognition and treatment. Although echocardiography has vastly improved our ability to diagnose this condition rapidly, a diagnostically adequate transthoracic echocardiographic study may not always be possible. Poor quality of images may result when certain physical characteristics of the patients (such as obesity) and chest diseases (such as emphysema) impede the transmission of ultrasound. With TEE, these difficulties are circumvented. The following cases illustrate the superior diagnostic ability of TEE over the conventional, transthoracic echocardiography in detecting vegetations located in unusual sites within the heart.

CASE REPORTS

CASE 1

A 74-year-old man was hospitalized with high fever. The exam-