CASE OF THE MONTH

ACUTE MYOCARDIAL INFARCTION IN AN UNSUSPECTING MALE

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PATIENT HISTORY

GE is a 54 year old Caucasian male who was in good health other than a history of gout. On the day of admission he had been exercising at a moderate level when he became aware of substernal chest pain. He had prompt relief of his pain upon cessation of exercise. He made two subsequent attempts to exercise but both had to be terminated because of the development of the same chest pain. At this point he sought medical attention in the emergency room where he was found to have evidence on EKG of an acute anterior myocardial infarction. He was immediately given 5 grains of ASA PO and started on tissue plasminogen activator (TPA) using a front loading protocol of 15 mgs IV bolus, followed by 5 mgs IV over the next thirty minutes, then 35 mgs over one hour. Heparin was started approximately two hours after the last dose of TPA.

The patient had been experiencing similar chest pains for one to two weeks with increasing frequency, usually with significant physical exertion. His discomfort had occasionally occurred at rest. Coronary risk factors included a mildly elevated total cholesterol and gout. His family history is negative for premature CAD and he is not hypertensive. He is not a smoker and current medications are 400 units of vitamin E daily.

Physical examination revealed a healthy appearing middle aged male who was complaining of mild chest discomfort but was not in distress. Blood pressure, heart rate, and respirations were normal. There was no corneal arcus or xanthelasma. His neck veins were flat and carotids had no bruits. Lungs were clear. Cardiac exam revealed a quiet precordium. First and second heart sounds were normal and there were no murmurs or gallops. His abdominal exam was negative and he had normal pulses in his extremities. The electrocardiogram revealed hyperacute ST elevation of eight-twelve mms in leads V2 through V6 with reciprocal ST depression in the inferior leads. Within ninety minutes of the initial bolus of TPA the ST elevation had resolved and his chest discomfort had subsided. Laboratory results obtained on admission and pre and post TPA are found in Table 1.

Cardiac catheterization was performed three days after admission. He was found to have a 95% eccentric ulcerated obstruction of the proximal segment of the left anterior descending coronary at the origin of the first septal perforator. The circumflex and right coronary arteries were free of obstructive disease. The left ventricular angiogram revealed normal wall motion and an ejection fraction of 60% indicating that early thrombolysis and myocardial reperfusion had thwarted any significant myocardial injury. This was reflected by only a modest cardiac enzyme rise with the CPK peaking at only 460 μ/L and MB fraction of 54 ng/ml.

Balloon angioplasty of the LAD was performed with a suboptimal result that required stenting with a Palmaz Schatz Stent to obtain a satisfactory unobstructed lumen.

Three days following the stenting of the LAD, he was discharged on Ticlid 250 mgs BID which was continued for four weeks after discharge. He was also started on Coumadin aiming for an INR of 2.0. Aspirin was continued at 5 grains daily. Metoprolol 50 mgs bid had been started shortly after admission and was continued.

Subsequent thallium treadmill testing was negative for reversible or fixed perfusion defects.

DISCUSSION

Coronary heart disease is known to affect over seven million individuals in the United States with a greater number being asymptomatic. Greater than five million people present to emergency rooms annually with chest pain, of which only 15% are actually experiencing a myocardial infarction. Early diagnosis followed by appropriate therapy is essential for positive patient outcomes.

This representative case is a situation where both clinical presentation and substantiating laboratory tests were indicative of myocardial infarction and demonstrated successful resolution of the clinical problems. ▲

		TpP	PF 1+2	TAT	FIB	TPA	PAI-1	D-Dimer
	Normal Range	0-3.5 µg/ml	0.5-3.0 nM/L	0.5-4.0 ng/ml	200-400 mg/dl	0.5-14 ng/ml	2.5-27.5 u/ml	0-100 ng/ml
TPA Infusion*	Admission	10	2.1	3.1	331	13.4	30.9	48
	TPA - 10 min.	4.2	1.5	3.2	118	18.8	29.6	662
	TPA - 1 hr.	3.5	6.2	34.7	138	2999	0	1275
	TPA - 2 hr.	3.8	2.9	9.3	125	180	0	2350
	TPA - 4 hr. (post-infusion)	2.6	1.8	6.2	114	31.5	15.8	1916
	TPA - 24 hr. (post-infusion)	2.3	1.2	2.6	227	24	17.9	142

*TPA infusion administered over 2 hours.

TpP = Thrombus Precursor Protein
PF 1+2 = Prothrombin Fragment 1+2
TAT = Thrombin/Antithrombin Complex
FIB = Fibrinogen
TPA = Tissue Plasminogen Activator
PAI-1 = Plasminogen Activator Inhibitor -1

Table 1.