ACUTE MI CASE STUDY

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- 2/22/00

PT PRESENTATION

- CC: “I feel like someone is squeezing my chest in a vise
- HPI: JF a 52 yo male , arrives by ambulance with crushing substernal CP. Onset 2 hrs PTA. Describes it as worst pain in life, radiating down L arm& jaw. Unrelieved by antacids, rest,3 SL NTG. Emesis X 2.

- PMH: HTN X 10 yrs
- FH: father died of stroke age 76, brother had MI age 54, brother with HTN
- SH: works 70-80 hrs/week as an executive, rarely exercises, 35 pk yr smoking, occas ETOH (1-2 beers/week)
- MEDS: Verapramil SR 240 mg qd X 4 yrs

- ALL: NKDA
- PE: GEN: WDWN man in acute distress, clutching chest
- VS: BP 175/100, P 105, RR 16, T 37.0 C, Ht 178 cm, Wt 88.5 kg
- HEART: tachycardic, nl S1,S2; no S3 or S4
- LUNGS: CTA
- EXT: diaphoretic, no edema

- LABS: NA+ 138, K+ 3.9, CL- 98, CO2 25, BUN 17, Cr 1.1, GLU 112, CK 180, 2% CK-MB
- ECG: 2-5 mm ST segment elevation in leads V1-V4

MI defn:

- Myocardial infarction is the result of a sudden interruption of blood to an area of the myocardium as a result of complete or partial occlusion of a coronary artery. The occlusion persists to compromise function and cause myocardium necrosis.

PATHOPHYSIOLOGY OF MI

- coronary artery disease (CAD) is primary underlying process
- CAD begins early in life
- fatty streaks deposit on coronary artery epithelium
- may progress to formation of atherosclerotic plaques (dependent on risk factors:HTN, DM, tob, hyperlipidemis)

- plaques eventually disrupt integrity & function of epithelium
- myocardial ischemia may occur due to the arteries’ narrowing
- coronary artery narrowing is NOT etiology
- 85% Mis caused by thrombus formation
- disruption of plaques initiates thrombotic process

- thrombotic process: activation of platelets, thrombin, and fibrin with subsequent thrombus formation occluding the vessel
- characterised by “wavefront” of ischemia progressing from endocardium to epicardium
- if blood flow not restored, myocardium dies within 3 hrs
- significant % salvageable up to 12-24 hrs

MYOCARDIAL ISCHEMIA

- occurs when there is inadequate O2 supply to meet myocardial demand (MVO2)
- primary determinant of O2 supply is coronary blood flow
- often occurs in CAD pts who increase demand with increased activity
- blood supply unable to meet increased demand

- ischemia occurs and localized area may become dysfunctional causing CP
- ischemia reversed when O2 demand is reduced therefore ischemic myocardium considered salvageable
- most likely precedes MI

- PRIMARY DIFFERENCE BETWEEN ISCHEMIA AND INFARCTION IS VIABLE MYOCARDIUM

FINDINGS C/W ACUTE MI

- crushing substernal CP radiating down L arm and to jaw, unrelieved by antacids, NTG, rest
- emesis, diaphoretic
- clutching chest
- elevated BP and HR
- ST segment elevation V1-V4
- LABS: CK 180 IU/L, 2 % CK-MB

RISK FACTORS

- HTN
- 35 pk yr smoking history
- 70-80 hr work week as an executive
- rarely exercises
- family history
- age 52
- male

GOALS OF PHARMACOTHERAPY

- minimize infarct size
- salvage ischemic myocardium
- prevent or minimize complications
- improve outcome

- drug therapy in early course of MI is directed at preventing/limiting early pump failure and maintaining patency of infarct artery

PHARMACOTHERAPEUTIC ALTERNATIVES

- IV MSO4
- NTG
- Lidocaine
- Beta-blockers
- Calcium channel blockers
- Thrombolytic
- Antithrombotic
- ACE inhibitors

NONPHARMACOLOGIC ALTERNATIVES

- Primary PTCA (percutaneous transluminal coronary angioplasty)
- supplemental O2
- activity restriction
- diet modification

PHARMACOTHERAPEUTIC PLAN

- IV MSO4 slowly 2-5 mg q 5-15 min as needed , dosage guided by BP. Persistent pain may need maintenance dose 4-8 mg q 4-6 hrs
- ASA 160-325 mg immediately on recognition of an MI
- NTG in 1st 24-48 hrs. IV preferred. 15 mcg bolus, followed by initial infusion rate 5-10 mcg/min.

- Beta-Blocker : Propanolol .1 mg/kg in 2-3 divided doses q 10 min , Metoprolol 5-10 mg in 2 divided doses q 5-10 min, Atenolol 5-10 mg in 2 divided doses q 5-10 min
- Thrombolytic: Streptokinase 1.5 million units/50 ml NS or D5W over 60 min, Altiplase/tPA: 15 mg IV bolus followed by .75 mg/kg infusion (not to exceed 50 mg) over 30 min followed by .5 mg/kg infusion (not to exceed 35 mg) over 60 min

- Heparin IV 75u/kg bolus followed by 1000 u/hr infusion at time tPA started. Maintain aPTT 1.5-2.0 times control for 48 hrs at least
- with Streptokinase do not give bolus start heparin when a PTT less than 2.0 times control but no earlier than 4 hrs after starting strept

- ACE inhibitor : Lisinopril 5 mg qd initially followed by 10 mg/d.

MONITORING EFFICACY & ADVERSE EFFECTS

- MSO4 : eval efficacy by pain relief. Monitor closely for hypotension, resp depression and allergic reactions
- NTG : eval by relief of pain. Monitor HR and BP closely. Eval ECG. Asssess for HA
- Beta-Blocker: Improvement of tachycardia and hypertension indicate efficacy. Reassess carefully HR and BP after each dose. Review ECG for AV block

- Thrombolytic: Assess for reperfusion by normalization of ST-segment on ECG, relief of CP, abrupt onset of arrhythmias (usually self-limiting) . Monitor for bleeding/stroke (increased with concurrent use of heparin and in hypertensive pt), hypotension, and allergic reaction (with streptokinase)

- Heparin : Monitor for bleeding. Monitor aPTT and maintain at 1.5-2.0 times control for 48 hrs and then switch to low dose subq until ambulatory
- ASA: Monitor for bleeding and GI effects

- ACE inhibitor: Eval efficacy by indication of improved hemodynamics, ie cardiac output. Monitor BP closely, signs/symptoms of worsening heart failure, renal function and K+ Observe for S/E: altered taste, cough, and diarrhea

CLINICAL COURSE

- 1 HR AFTER THROMBOLYTIC INFUSION: CP resolved; ST elevation to baseline;peak CT 1465 and 24 % CK-MB
- DAY 5: 90% occlusion LAD coronary artery; normal circumflex branch & R coronary artery, EF 45%
- DAY 6: no CP,ECG changes, or areas of reversible ischemia on exercise thallium test
- DAY 7: D/Cd home

DISCHARGE MEDICATIONS

- ASA 160-325 mg EC qd for life
- Atenolol 100 mg qd
- NTG SL prn
- Lisinopril 10 mg qd

INFORMATION FOR PT

- diet: low fat, low salt, lipid mgt
- guidelines for resumption of activities (including sexual)
- guidelines for resumption of work
- smoking cessation programs
- HTN mgt

- educational materials re: nature of disease
- med list with S/E and cautions
- info on cardiac rehab programs
- stress mgt resources

ADDITIONAL RECOMMENDATIONS: 6 wks later

- obtain lipid profile
- family education re: prevention and evaluation
- stress test

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