

CASE STUDY 16 - UNSTABLE ANGINA

CHIEF COMPLAINT and SIGNIFICANT HISTORY:

38 y/o MALE presents with sensation of exertional CHEST and NECK PAIN, described as "burning." Patient states symptoms also occur when he is under emotional duress. Symptoms have been occurring intermittently for approx. 2-3 weeks.

RISK FACTOR PROFILE:

-  **HYPERTENSION**
-  **DIABETES x 5 YEARS**

PHYSICAL EXAM: Pt. ASYMPTOMATIC at time of exam. SKIN WARM, DRY, COLOR NORMAL, PERLA, LUNGS= CLEAR, HS NORMAL S1, S2, NO ANKLE EDEMA.

VITAL SIGNS: BP: 144/92 P: 78 R: 16 SAO2: 100% on room air

LABS: TROPONIN: <.04

QUADRAD OF ACS CHECKLIST

☒ SYMPTOMS of ACS

- ☒ **TYPICAL ACS - eg:**
- ☐ **ATYPICAL ACS - eg:**

☐ ECG ABNORMALITIES

- ☐ **ST ELEVATION** (J POINT plus 40 ms)
- ☐ **HYPERACUTE T WAVES** - and/or -
- ☐ **NEW or PRESUMABLY NEW LBBB**
- ☐ **ST DEPRESSION** (>0.5 mm @ J POINT) and/or
- ☐ **INVERTED or BIPHASIC T WAVES** and/or
- ☐ **DYNAMIC ST SEGMENT and/or T WAVE CHANGES** IN SERIAL EKGs

☐ RISK FACTORS - 3 or more

- ☐ **FAMILY HISTORY**
- ☒ **DIABETES**
- ☐ **↑ LDL and/or ↓ HDL**
- ☐ **SMOKING**
- ☐ **AGE: 65 or MORE**
- ☒ **HYPERTENSION**

☐ CARDIAC MARKERS

- ☐ **ELEVATED TROPONIN and/or CK/MB**

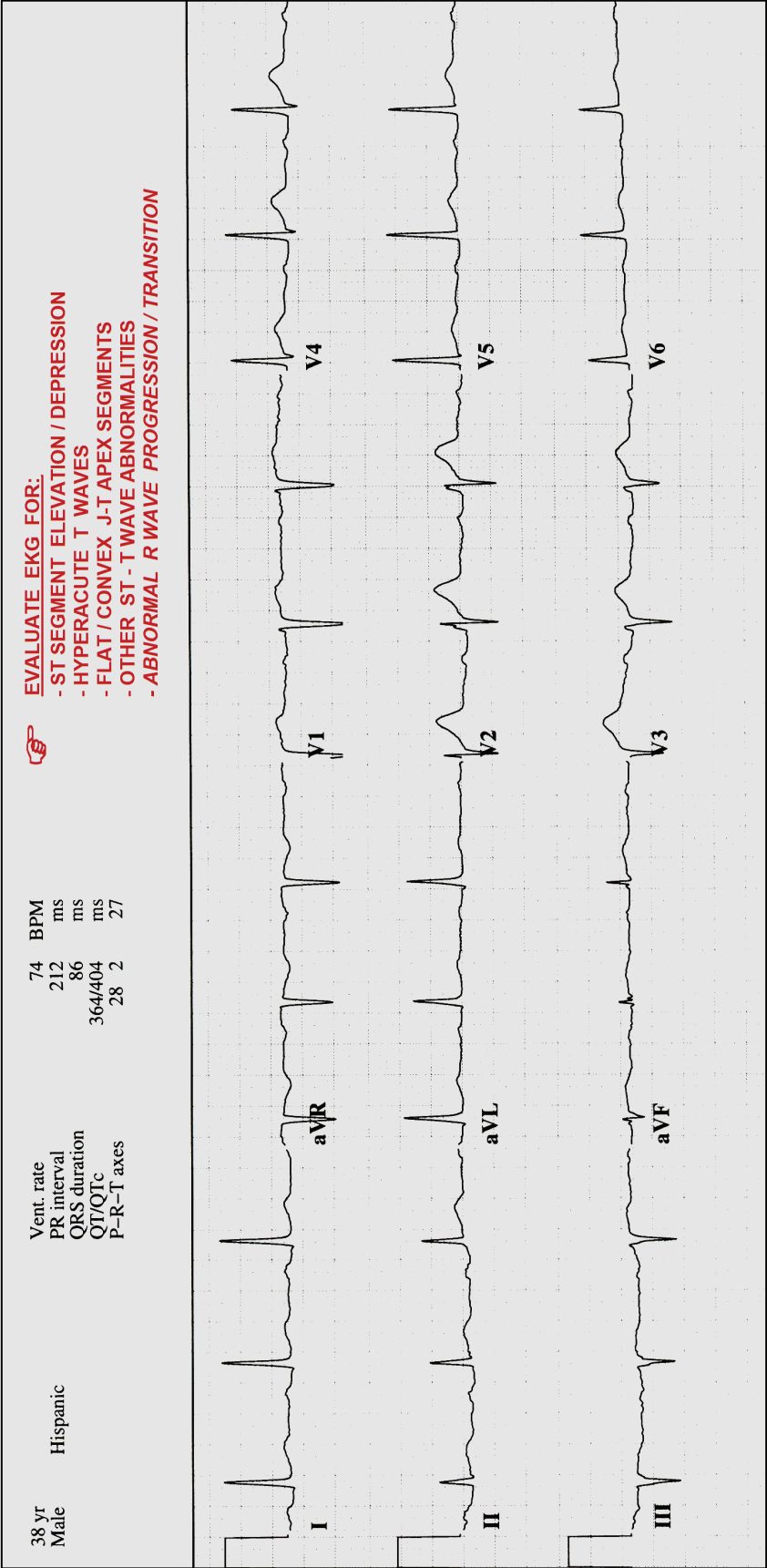
☐ TOTAL

This patient complained of exertional chest and neck pain, which he described as a "burning sensation," which did not radiate to shoulders, jaw or arms. His first episode was approximately three weeks ago, and occurred during physical exertion. In the ensuing weeks, episodes have become more frequent, and have been provoked by emotional distress. For the past week, he states the chest pain has come on at rest, and has awakened him in the morning on two occasions.

Since his past medical history included hypertension and insulin-controlled diabetes mellitus, he feared his new onset of chest discomfort may be heart-related.

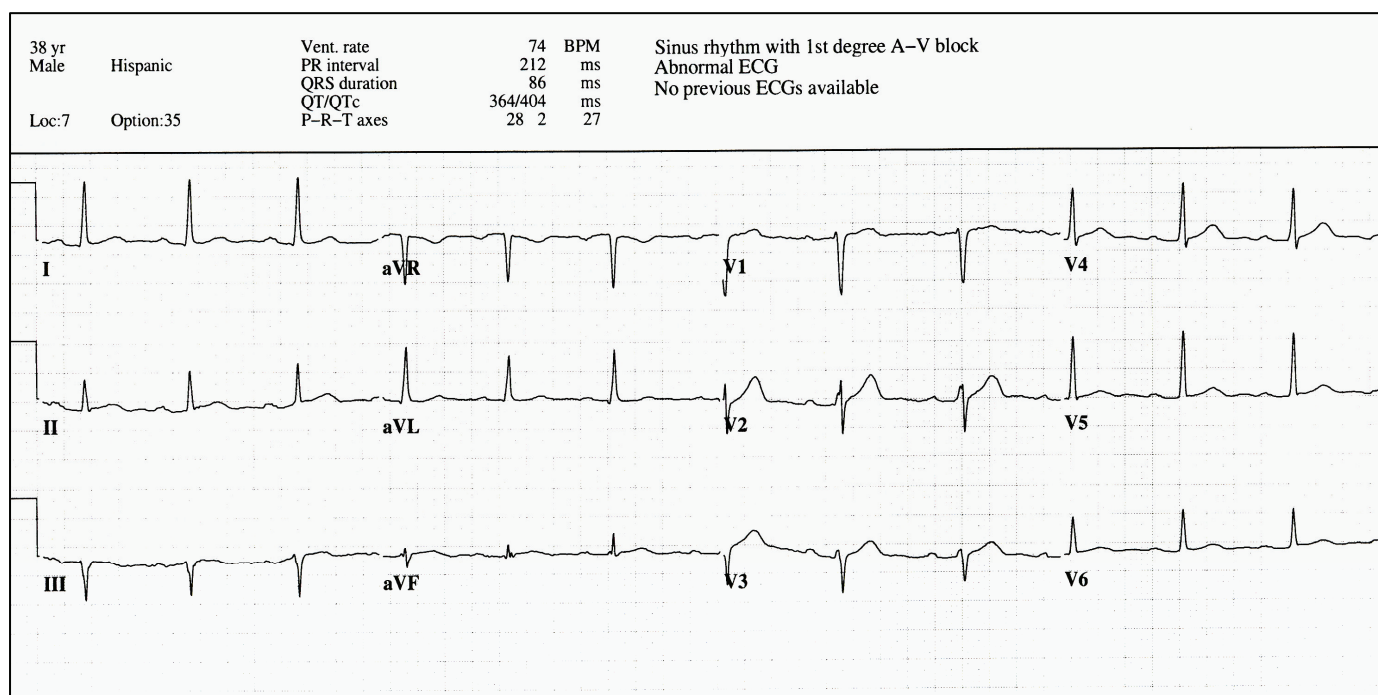
During exercise stress testing, ST segment elevation in leads II, III, and aVF were noted, along with the onset of his symptoms. At this point, the stress test was aborted, with resolution of ST elevation and symptoms.

He subsequently was referred for an urgent cardiac catheterization, with a diagnosis of "unstable angina."



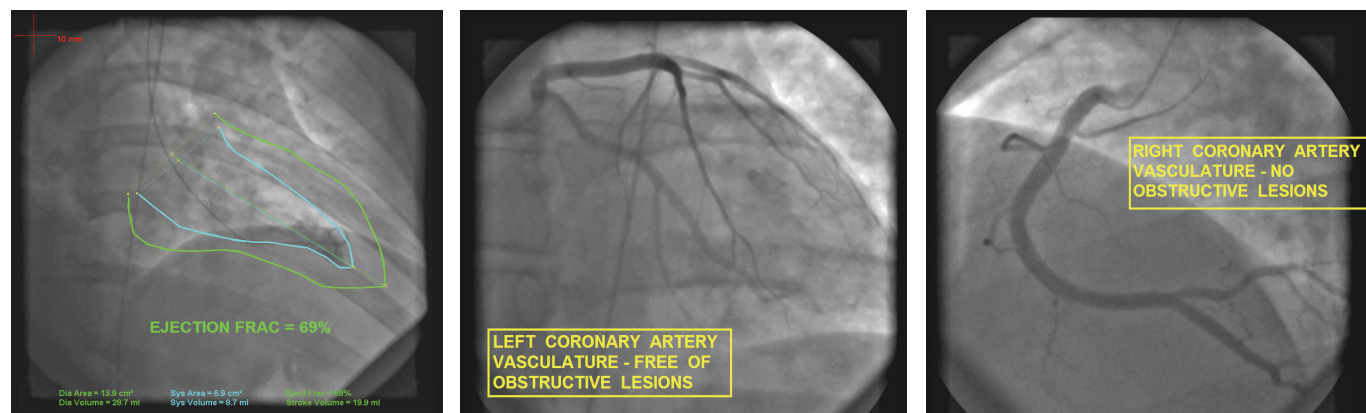
CASE STUDY QUESTIONS:

NOTE LEADS WITH ST ELEVATION:	NOTE LEADS WITH ST DEPRESSION:
WHAT IS THE SUSPECTED DIAGNOSIS ?	
WHAT IS THE "CULPRIT ARTERY" -- if applicable ?	
LIST ANY CRITICAL STRUCTURES COMPROMISED:	LIST ANY POTENTIAL COMPLICATIONS:



As noted by the ECG computer diagnosis, first degree heart block is present, with a P-R interval of 212ms. Other than a slight R wave progression abnormality in lead V3, no other abnormalities are noted.

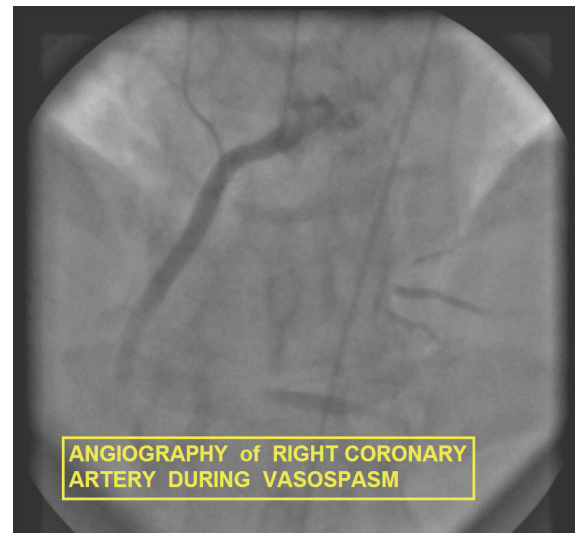
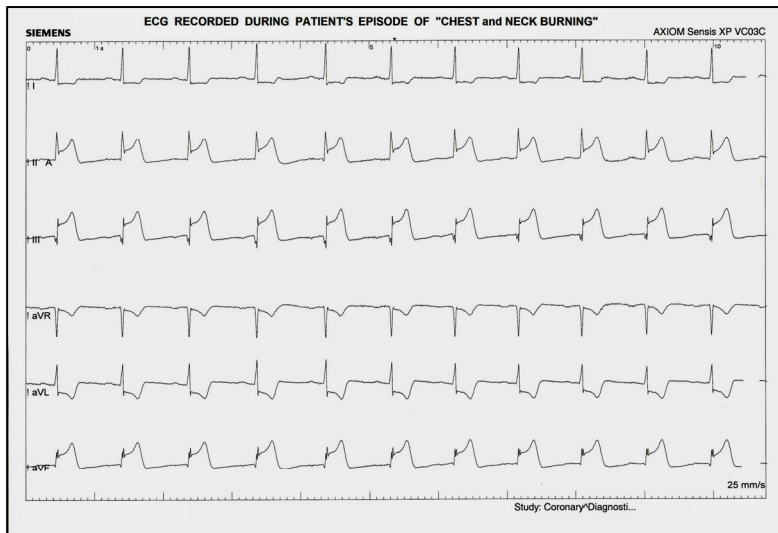
He was taken to the cardiac cath lab, where the following images were obtained:



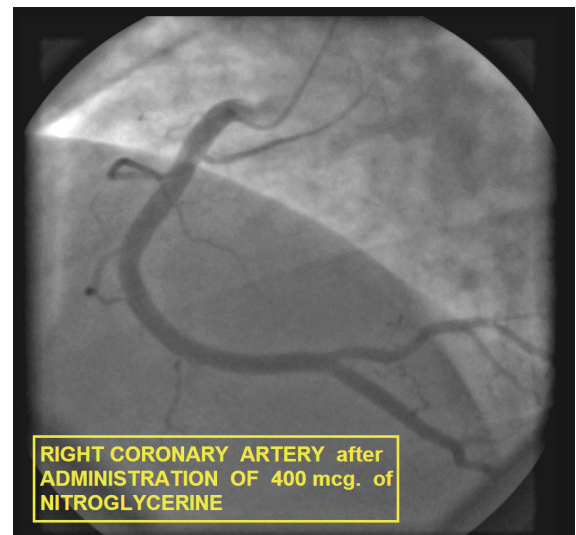
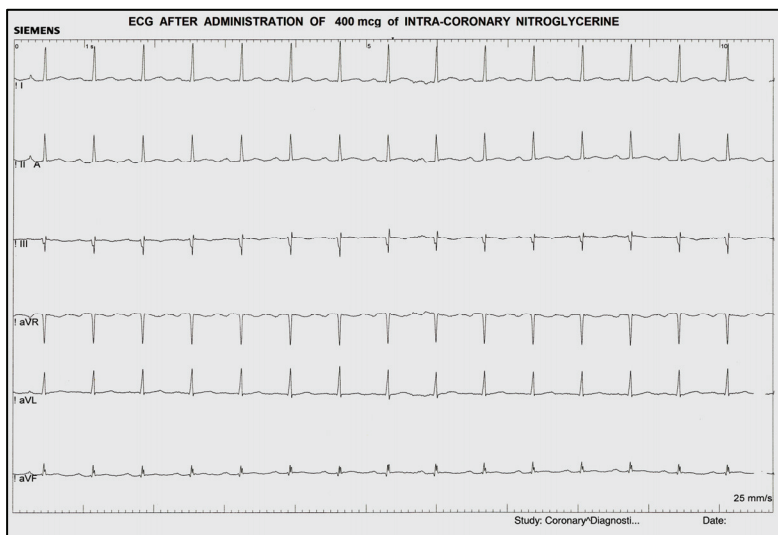
His left ventricular angiogram, seen to the above left, is normal, and shows no sign of injury. His ejection fraction is measured at 69%. Reference for normal range is 55 – 70%.

His left coronary artery system (above middle) shows diffuse, non-obstructive LAD disease in the mid to distal segment. The right coronary artery (above right) is remarkably free of obstructive coronary artery disease.

Within seconds after the above right image was obtained, the patient began complaining of a severe “burning” pain in his chest and neck. The 6 lead ECG tracing at the top of the next page was recorded during the patient’s episode of chest pain. ST segment elevation is noted in leads II, III, and aVF, which is consistent with occlusion of a dominant right coronary artery. Repeat angiography of the right coronary artery revealed severe mid-segment vasospasm, as seen in top right image on the next page.



Next, 400 micrograms of intra-coronary nitroglycerine was administered, which effectively resolved the arterial vasospasm. The ECG and angiography below were taken just after abatement of the patient's symptoms:



The patient was diagnosed with "Prinzmetal's Variant Angina" secondary to coronary artery vasospasm, and was placed on calcium channel blocker and nitrate therapy.

Prinzmetal or variant angina is caused by focal coronary artery vasospasm, and was first described by Myron Prinzmetal in 1959 as a syndrome of episodic chest pain that comes on at rest with ST segment elevation.⁶⁸ Prinzmetal angina is classified as unstable angina due to its unpredictability⁶⁹, and has been associated with myocardial infarction, ventricular dysrhythmias and cardiac arrest. The primary mechanism of vasospasm is hypercontraction of vascular smooth muscle cells. Variant angina is not an indicator of CAD; many patients are free of atherosclerotic plaque. Some factors known to provoke coronary artery vasospasm include: vasoconstrictor medications, stimulants such as cocaine, ephedrine and amphetamines, emotional duress, exposure to cold and alcohol withdraw.

Typical Prinzmetal's variant angina occurs at rest, in the early hours of the morning. The pain is often described as severe chest tightness or pressure. Variant angina is usually treated with and responds well to calcium channel blockers and nitrates.⁷⁰

⁶⁸ Prinzmetal et al, *Am J Med*. 1959;27:375-388.

⁶⁹ National Institutes of Health, Library of Medicine, www.NIH.gov

⁷⁰ National Institutes of Health, Library of Medicine, www.NIH.gov