

Unstable angina prior to presentation with STEMI: insights from a 'Real-world' series of patients undergoing PPCI - Sall H - University Hospitals of Burton

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Abstract

Hypothesis generating study describing STEMI presentation, influence of medical contact; relationship between presentation and infarct size.

51 patients undergoing PPCI completed questionnaires. Peak CK level within 72hours was used as an infarct size surrogate. Student's t test compared continuous variables between groups. Univariate logistic regression used to test associations. Statistical analysis by SISA Binomial. Pre-infarction unstable angina pectoris (UAP) can be considered ischemic preconditioning. The aim of this study was to compare short and long term outcomes in patients with or without pre-infarction UAP and ST elevation myocardial infarction (STEMI) treated with primary percutaneous coronary intervention (PCI).

Median age was 64(41-85) years;45male(88.2%). 52.9%(27/51)had anterior MI. 21.6%lived alone, 43.1%were employed, 27.4%current smokers. 68.6%experienced symptom onset at home, 15.9%whilst working. 37.2%experienced chest pain the week previous, 25.4%more than 48hrs prior. Of these 62%at rest, 49%crescendo nature, and 21%on exertion. Most frequently associated symptom;left upper limb pain(51%)and jaw/throat ache(42%). 41.2%thought symptoms were indigestion. 35.3%consulted a medical professional(primary care;61%) with symptoms within one month prior. 15.6%report being reassured and given an alternative diagnosis. 1 patient was referred to rapid access chest pain clinic but experienced STEMI prior. 9.8%self-presented to ED, the remainder presenting via paramedic PPCI pathway.

The levels of NT-proBNP were significantly higher on admission and after 24 hours in the UAP group. Left ventricular functions according to invasive and echocardiographic parameters were entirely

comparable at hospitalization and after 12 months. No differences were found in severity index of acute heart failure during hospitalization. The incidence of major acute coronary events during follow-up was comparable for the groups.

No relationship found between peak CK and living alone, location at time of symptom onset or self-presentation to ED vs PPCI pathway(2307vs1691 U/L, $p=0.178$). Patient understanding of diagnosis was rated as good(92%).

In this 'real-world' series more than one third of patients experienced unstable angina within 4 weeks of STEMI and one in 7 were inappropriately reassured. There may be a persistent unmet educational need around diagnosis of ACS in the general population and primary care. Further research is required to describe the presentation of ACS including STEMI: this may inform future models for specialist assessment of chest pain. In patients with STEMI treated with primary PCI, pre-infarction UAP has no beneficial clinical effect during hospitalization or during long-term follow-up. Acute coronary syndromes (ACSs) represent a life-threatening range of clinical conditions that are almost always associated with the rupture of an atherosclerotic plaque and partial or complete thrombosis of the infarct-related artery. Platelet aggregation, induced by plaque rupture, is an important contributor to the generation of atherothrombotic events. Dual antiplatelet therapy (DAPT) consisting of aspirin and a P2Y₁₂ receptor inhibitor is the recommended treatment following an ACS. Guidelines include maintaining a long-term DAPT course for one year after an ACS event. Early discontinuation of DAPT has been associated with adverse outcomes. A retrospective observational cohort study from the UK assessing clopidogrel therapy persistence in a population of 4650 patients

discharged after acute myocardial infarction (MI) found that premature discontinuation of clopidogrel during the first 12 months of treatment was associated with a significant increase in the risk of death or recurrent infarction.

Unstable angina belongs to the spectrum of clinical presentations referred to collectively as acute coronary syndromes (ACSs), which also includes ST-segment elevation myocardial infarction (STEMI) and non-STEMI (NSTEMI). [1, 2] Unstable angina is considered to be an ACS in which there is myocardial ischemia without detectable myocardial necrosis (ie, cardiac biomarkers of myocardial necrosis —such as creatine kinase MB isozyme, troponin, myoglobin— are not released into the circulation). See the image below.

The diagnosis of unstable angina and non-STEMI is predominantly based on the ECG and cardiac enzymes. Physical examination, as previously described, is non-specific. The ECG tracing can have multiple abnormalities, but, by definition, there is no ST segment elevation.

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During an unstable angina event: You may get heparin (or another blood thinner) and nitroglycerin (under the tongue or through an IV). Other treatments may include medicines to control blood pressure, anxiety, abnormal heart rhythms, and cholesterol (such as a statin drug).

The most common form of nitrate used to treat angina is nitroglycerin tablets, which you put under your tongue. Aspirin. Aspirin reduces the ability of your blood to clot, making it easier for blood to flow through narrowed heart arteries. Preventing blood clots can also reduce your risk of a heart attack.

Unstable angina may not respond to rest or nitroglycerin. It needs urgent attention. Usually lasts 5 minutes; rarely more than 15 minutes