Myocardial Infarction and Angina Pectoris (Project)

Maha Mohamed Abo El-Mattey (Student)

School of Nursing, Kaser Elainey, Egypt

Abstract: Survivors of myocardial infarction have a hypofibrino lytic state, characterized by increased basal plasminogen activator inhibitor (PAI-I) activity combined with low tissue plasminogen activator (tPA) activity after venousocclusion test', On the other hand, tPA antigen concentrations may well prove to be high!", Similar findings have been observed in patients with angina pectoris l>-SJ. In one report, increased plasma PAI-I activity was found to be an independent risk factor for reinfarction and cardiac death among young survivors of Mil. The fibrinolytic key components tPA and PAI-I, are thus to be considered as novel cardiovascular risk factors. The present study was designed to evaluate tPA antigen and PAI-I as predictors for cardiovascular events; myocardial infarction, stroke and cardiovascular death, com• pared with previously established risk factors, in patients with severe angina pectoris and angiographically verified coronary artery disease.
[Maha Mohamed Abo El-Mattey. **Myocardial Infarction and Angina Pectoris (Project).** *Biomedicine and Nursing* 2018;4(4): 1-5]. ISSN 2379-8211 (print); ISSN 2379-8203 (online). http://www.nbmedicine.org. 1. doi:10.7537/marsbnj040418.01.

Keywords: Myocardial Infarction; Angina; Pectoris; Project

Introduction

The advent of beta-receptor blocking agents in the management of angina pectoris has been a major advance in a field where previously the profusion of long-acting coronary vasodilators used was a testimony to their ineffectiveness. Pronethalol was the first successful drug in the treatment of angina (Dornhorst and Robinson, 1962; Alleyne et al., 1963), and was soon superseded by propranolol, which bas a therapeutic potency tenfold that of pronethalol and is free of the untoward sideffects of that compound (Black et al., 1964). More recently a newer compound has been produced, verapamil (Cordilox, iproveratril, Isoptin), which on the basis of experimental work by Hass (1964) was introduced as a new beta-receptor blocking agent, and it was claimed that, unlike propranolol, yerapamil docs not induce either coronary artery vasoconstriction or bronchoconstriction (Haas, 1964). These experimental results have, however, been challenged (Fitzgerald and Barrett, 1967; Wilkinson, 1967; Grant et al., 1968), and in the present state of knowledge it cannot be convincingly maintained that verapamil is indeed a beta-receptor blocking agent.

However, some preliminary clinical studies of the use of verapamil in angina have shown its efficacy but these have been uncontrolled and assessment has been largely subjective, though another study by Wette et al. (1966) has shown improvement in the ischaemic patterns in the electrocardiogram after longterm treatment with verapamil. Provided verapamil has no deleterious effects on the heart or other organs then the exact mechanism of action in angina, whether this involves beta• receptor blockade or direct depression of myocardial contractility, is of less importance than whether the drug is an effective agent in angina pectoris. It was with these considerations in mind that it was decided to undertake a controlled double blind evaluation of verapamil in the treatment of angina. Basing the assessment mainly on the objective criteria provided by exercise tolerance tests, and at the same time a comparison was made in the same patients between verapamil and propranolol, already widely used in the treatment of angina.

CORONARY thrombosis is now generally recognized as the precipitating event in the transition from stable to acute ischemic heart disease, manifested by unstable angina, acute myocardial infarction, and sudden death from coronary causes. Besides local stimuli such as disruption of plaques, systemic thrombogenic factors may contribute to the occurrence, extent, and persistence of coronary thrombosis and its clinical sequelae. These factors include abnormalities of blood flow, platelet hyperreactivity, defective fibrinolysis, and increased concentrations of hemostatic proteins, specifically fibrinogen and factor VII.

Thus, with the use of appropriate laboratory tests, it may be possible to detect a thrombogenic state and thus identify patients at increased risk for cardiovascular disease.

Support for this hypothesis comes mainly from pro- spective studies of healthy subjects, which have demon- strated a direct and independent association between plasma fibrinogen concentrations and the risk of coro- nary events. However, data on patients with known coronary artery disease are sparse and come from small cohort studies of patients with angina pectorisor those who have had a first myocardial Infarction Patients with chest pain in the absence of obstructive coronary artery disease (CAD) remain a challenge. More than half of women with stable chest

pain undergoing coronary angiography G) are found to have no obstructive CAD, while this is true for only one-third of men. Until recently, the prognosis was thought to be benign and many of these patients have been offered little more than reassurance that they do not have serious heart disease. However, the perception of the benign patients with chest pain in the absence of obstructive coronary artery disease (CAD) remain a challenge. More than half of women with stable chest pain undergoing coronary angiography (CAG) are found to have no obstructive CAD, while this is true for only one-third of men. Until recently, the prognosis was thought to be benign and many of these patients have been offered little more than reassurance that they do not have serious heart disease. However, the perception of the benign nature of the condition in women has been challenged with evidence from the Women's Ischemia Syndrome Evaluation (WISE) study showing that women with symptoms and signs suggestive of myocardial ischaemia but without obstructive CAD are at elevated risk for cardiovascular events. Furthermore, these patients often continue to have chest pain leading to anxiety, limited physical capacity, and reduction in quality of life and making them more likely to be readmitted to repeated procedures and medical assessment. Some patients with no obstructive CAD might have chest pain due to cardiac diseases other than ischaemic heart disease due to coronary atherosclerosis. In the WISE study, it was hypothesized that the increased risk of cardiovascular outcomes was due to endothelial dysfunction not seen by traditional CAG.

This is in line with other studies that have addressed the long-term prognostic value of endothelial function testing in patients with no obstructive CAD and demonstrated that endothelial dysfunction is associated with increased numbers of adverse cardiovascular events. Importantly, however, the WISE study only enrolled women. Therefore, it is unknown whether these results are gender-specific and whether women suspected of myocardial ischaemia but without obstructive CAD might differ from men in terms of prognosis.

We investigated the prognostic implications of cardiac symptoms of stable angina pectoris in patients with no obstructive CAD in a cohort of women and men referred for CAG and compared them with a reference sample from the background population and with patients with obstructive CAD.

The major independent role played by anxiety and severe psychosocial problems (especially family ones) is demonstrated by this multivariate analysis of a five year prospective study of the development of new angina pectoris among almost 10,000 adult men (average annual incidence= 5.7/1,000).

The Independent effect of these two variables is considerably augmented by the other significant risk factors of age, total serum cholesterol, systolic or diastolic blood pressure, certain electrocardlographlc abnormalities and diabetes mellltus. The presence of all seven risk factors (at a high level) increases the probability of angina pectoris developing within five years to 289/1,000 from 14/1,000, when these factors are low or absent.

The wife's love and support is an important balancing factor, which apparently reduces the risk of angina pectoris even in the presence of high risk factors. The implications of these findings to the pathophysiology and prevention of angina are stressed.

Angina pectoris, known since antiquity, has been the subject of many anecdotal, clinical and research publications. Despite this, there are still many puzzling features related to its physiology, pathology, etiology and prognosis.

In an attempt to elucidate some of the factors associated with the development of angina pectoris, 10,000 adult Israeli men, aged 40 years and over, were followed intensively for five years. The incidence over this period and the univariate analysis has been pre• viously reported [1]. Of the more than 100 variables tested, those found to be associated in the univariate analysis with the develop• ment of angina pectoris were (corresponding to single-test $p < 0.01$) as follows [1): Sociodemographic and genetic-those born in Southeastern Europe, age, blood group A 18Jka- (Jka- = Kidd negative). Clinical-blood pressure (systolic and diastolic), intermittent claudication, diabetes mellitus (and casual blood glucose), nonspecific T waves in the resting electrocardiogram, total serum cholesterol, cholesterol in beta-lipoprotein. Psychosocial-anxiety, severe problems of whatever nature, geographic (country) mobility. In addition, the following variables were significant at $p < 0.05$ level: overweight and peptic (duodenal) ulcer.

Previous meta-analyses of randomised trials have shown that antiplatelet therapy prevents serious vascu-lar events, arterial occlusion, and venous thromboembolism3 among a wide range of patients at high risk f occlusive vascular events. The proportional reduction in serious vascular events (non-fatal myocardial infarction, non-fatal stroke, or death from a vascular cause) was about one quarter in a wide range of high risk patients, irrespective of why the risk was high and irrespective of age, sex, blood pressure, or history of diabetes.

The previous meta-analyses, however, left some important clinical questions unanswered. For instance, although long term ntiplatelet therapy was shown to be of substantial benefit after ischaemic stroke, it was not known whether antiplatelet drugs were of net benefit as an immediate treatment in the acute phase of such

strokes. There was also some uncertainty about whether antiplatelet therapy was of net benefit in patients with chronic conditions such as atrial fibrillation, stable angina, and atherosclerotic periph-eral arterial disease that had been less extensively studied. Daily doses of at least 75 mg of aspirin had been shown to be effective in long term use, but theoretical advantages had been proposed for lower doses.

The previous meta-analyses included only those trials that were available in 1990, and since then there have been many additional trials of aspirin at various doses and of other antiplatelet drugs.

There have also been trials of the effects of adding to aspirin another antiplatelet drug with a different mechanism of action.

In addition, although certain anticoagulant regimens were known to be effective for particular high risk patients in the absence of antiplatelet therapy, it was not known whether the addition of anticoagulants to antiplatelets would provide additional protection. We have therefore updated previous meta-analyses to include studies available by September 1997. This paper summarises the updated results from the trials of antiplatelet drugs among high risk patients.

Myocardial infarction (MI) can be recognized by clinical features, including lectrocardiographic (ECG) findings, elevated values of biochemical markers (biomarkers) of myocardial necrosis, and by imaging, or may be defined by pathology (Box 1). It is a major cause of death and disability worldwide. MI may be the first manifestation of coronary artery disease (CAD) or it may occur, repeatedly, in patients with established disease. Information on MI rates can provide useful information regarding the burden of CAD within and across populations, especially if standardized data are collected in a manner that distinguishes between incident and recurrent events. From the epidemiological point of view, the incidence of MI in a population can be used as a proxy for the prevalence of CAD in that population. The term ‘myocardial infarction’ may have major psychological and legal implications for the individual and society. It is an ndicator of one of the leading health problems in the world and it is an outcome measure in clinical trials, observational studies and quality assurance programs. These studies and programs require a precise and consistent definition of MI.

In the past, a general consensus existed for the clinical syndrome designated as MI. In studies of disease prevalence, the World Health Organization (WHO) defined MI from symptoms, ECG abnormalities and cardiac enzymes. However, the development of ever more sensitive and myocardial tissue-specific cardiac biomarkers and more sensitive imaging techniques now allows for detection of very small amounts of myo cardial injury or necrosis. Additionally, the management of patients with MI has significantly improved, resulting in less myocardial injury and necrosis, in spite of a similar clinical presentation. Moreover, it appears necessary development of even more sensitive assays for markers of myocardial necrosis mandates further revision, particularly when such necrosis occurs in the setting of the critically ill, after percutaneous coronary procedures or after cardiac surgery. The Third Global MI Task Force has continued the Joint ESC/ACCF/AHA/WHF efforts by integrating these insights and new data into the current document, which now recognizes that very small amounts of myocardial injury or necrosis can be detected by biochemical markers and/or imaging.

The management of acute myocardial infarction continues to undergo major changes. Good practice should be based on sound evidence derived from well-conducted clinical trials. Because of the great number of trials on new treatments performed in recent years and because of new diagnostic tests, the European Society of Cardiology decided that it was opportune to upgrade the 1996 guidelines and appointed a Task Force. It must be recognized, that even when excellent clinical trials have been undertaken, their results are open to interpretation and that treatment options may be limited by resources. Indeed, cost-effectiveness is becoming an increasingly important issue when deciding upon therapeutic strategies.

The definition of acute myocardial infarction

Myocardial infarction can be defined from a number of different perspectives related to clinical, electrocardiographic (ECG), biochemical and pathologic characteristics. It is accepted that the term myocardial infarction reflects death of cardiac myocytes caused by prolonged ischaemia.

The ECG may show signs of myocardial ischaemia, specifically ST and T changes, as well as signs of myocardial necrosis, specifically changes in the QRS pattern. A working definition for acute evolving myocardial infarction in the presence of clinically appropriate symptoms has been established as (1) patients with ST-segment elevation, i.e. new ST-segment elevation at the J point with the cut-off points ≥0.2 mV in V1 through V3 and ≥0.1 mV in other leads, or patients without ST-segment elevation, i.e. ST-segment depression or T wave abnormalities. Clinically established myocardial infarction may be defined by any Q wave in leads V1 through V3, or Q wave ≥0.03 s in leads I, II, aVL, aVF, V4, V5 or V6.

Myocardial infarction can be recognized when blood levels of biomarkers are increased in the clinical setting of acute myocardial ischaemia. The preferred biomarker for myocardial damage is cardiac troponin

(I or T) which has nearly absolute myocardial tissue specificity, as well as high sensitivity. The best alternative is CK-MB mass, which is less tissue-specific than cardiac troponin but its clinical specificity for irreversible injury is more robust. An increased value of cardiac troponin or CK-MB is defined as one that exceeds the 99th percentile of a reference population.

The present guidelines pertain to patients presenting with ischaemic symptoms and persistent ST-segment elevation on the ECG. The great majority of these patients will show a typical rise of biomarkers of myocardial necrosis and progress to Q-wave myocardial infarction. Separate guidelines2 have been developed by another Task Force of the European Society of Cardiology for patients presenting with ischaemic symptoms but without persistent ST-segment elevation.

Conclusion

In setting out these new guidelines, the Task Force has attempted to classify the usefulness or efficacy of the recommended routine treatments and the level of evidence on which these recommendations are based. The usefulness or efficacy of a recommended treatment will be presented.

The strength of evidence will be ranked according to three levels: level A, data derived from at least two randomized clinical trials; level B, data derived from a single randomized clinical trial and/or metaanalysis or from non-randomized studies; level C, consensus opinion of the experts based on trials and clinical experience. As always with guidelines, they are not prescriptive. Patients vary so much from one another that individual care is paramount and there is still an important place for clinical judgment, experience and common sense.

References

1. The Joint European Society of Cardiology/American College of Cardiology Committee. Myocardial infarction redefined—A consensus document of the Joint European Society of Cardiology/American College of Cardiology for the redefinition of myocardial infarction. Eur Heart J 2000;21:1502–13.
2. Grijseels EW, Deckers JW, Hoes AW et al. Prehospital triage of patients with suspected acute myocardial infarction. Evaluation of previously developed algorithms and new proposals. Eur Heart J 1995; 16:325–32.
3. Hauser AM, Gangadharan V, Ramos RG et al. Sequence of mechanical, electrocardiographic and clinical effects of repeated coronary arterial occlusion in human beings: echocardiographic observation.
4. Bertrand ME, Simoons ML, Fox KA et al. Management of acute coronary syndromes: acute coronary syndromes without persistent ST segment elevation. Recommendations of the Task Force of the European Society of Cardiology. Eur Heart J 2000;221:1406–32. During coronary angioplasty. J Am Coll Cardiol 1985;5:193–7.
5. Davies MJ. The pathophysiology of acute coronary syndromes. Heart 2000;83:361–6.
6. Falk E, Shah PK, Fuster V. Coronary plaque disruption. Circulation 1995;92:657–71. 18. Lengyel M. The role of transesophageal echocardiography in the management of patients with acute and chronic pulmonary thromboembolism. Echocardiography 1995; 12:359–66.
7. Tatum JL, Jesse RL, Kontos MC et al. Comprehensive strategy for the evaluation and triage of the chest pain patient. Ann Emerg Med 1997;29:116–25.
8. Heller GV, Stowers SA, Hendel RC et al. Clinical value of acute rest technetium-99m tetrofosmin tomographic myocardial perfusion imaging in patients with acute chest pain and nondiagnostic electrocardiograms. J Am Coll Cardiol.
9. Alderman EL, Corley SD, Fisher LD et al. Five-year angiographic follow-up of factors associated with progression of coronary artery disease in the Coronary Artery Surgery Study (CASS). CASS Participating Investigators and Staff. J Am Coll Cardiol 1993; 22:1141–54. 1998;31:1011–7.
10. Falk E. Unstable angina with fatal outcome: dynamic coronary thrombosis leading to infarction and/or sudden death. Autopsy evidence of recurrent mural thrombosis with peripheral embolization culminating in total vascular occlusion. Circulation 1985;71:699–708.
11. Topol EJ, Yadav JS. Recognition of the importance of embolization in atherosclerotic vascular disease. Circulation 2000;101:570–80.
12. Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2000; 102: I-22–I-59.
13. Guidelines 2000 for cardiopulmonary resuscitation and emergency cardiovascular care. Circulation 2000; 102: I-82–I-166.
14. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomized trials of more than 1000 patients. Lancet 1994; 343:311–22.
15. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Randomised trial of intravenous streptokinase, oral aspirin, both, or

neither among 17,187 cases of suspected acute myocardial infarction: ISIS-2. Lancet 1988; ii:349–60.
16. ISIS-3 (Third International Study of Infarct Survival) Collaborative Group. ISIS-3: a randomised comparison of streptokinase vs tissue plasminogen activator vs anistreplase and of aspirin plus heparin vs aspirin alone among 41,299 cases of suspected acute myocardial infarction. Lancet 1992;339:753–70.
17. Armstrong A, Duncan B, Oliver MF et al. Natural history of acute coronary heart attacks. A community study. Br Heart J 1972;34:67–80.
18. Tunstall-Pedoe H, Kuulasmaa K, Mahonen M et al. Contribution of trends in survival and coronary-event rates to changes in coronary heart disease mortality: 10-year results from 37 WHO MONICA project populations. Monitoring trends and determinants in cardiovascular disease. Lancet1999;353:1547–57.
19. Norris RM. Fatality outside hospital from acute coronary events in three British districts, 1994–5. United Kingdom Heart Attack Study Collaborative Group. BMJ 1998; 316:1065–70.
20. Norris RM, Caughey DE, Mercer CJ et al. Prognosis after myocardial infarction. Six-year follow-up. Br Heart J 1974; 36:786–90.
21. de Vreede JJ, Gorgels AP, Verstraaten GM et al. Did prognosis after acute myocardial infarction change during the past 30 years? A meta-analysis. J Am Coll Cardiol 1991; 18:698–706.
22. Hasai D, Begar S, Wallentin L et al. A prospective survey of the characteristics, treatments and outcomes of patients with acute coronary syndromes in Europe and the Mediterranean basin. The Euro Heart Survey of Acute Coronary Syndromes (Euro Heart Survey ACS). Eur Heart J 2002; 15:1190–201.
23. Lee KL, Woodlief LH, Topol EJ et al. Predictors of 30-day mortality in the era of reperfusion for acute myocardial infarction. Results from an international trial of 41,021venous thrombolytic treatment in acute myocardial infarction. Lancet 1986;1:397–402.
24. The International Study Group. In-hospital mortality and clinical course of 20,891 patients with suspected acute myocardial infarction randomised between alteplase and streptokinase with or without heparin. Lancet 1990; 336:71–5.
25. The GUSTO Investigators. An international randomized trial comparing four thrombolytic strategies for acute myocardial infarction. N Engl J Med 1993; 329:673–82.
26. Neuhaus KL, Feuerer W, Jeep-Tebbe S et al. Improved patients. GUSTO-I Investigators. Circulation 1995; 91:1659–68.
27. Adams J, Trent R, Rawles J, on behalf of the GREAT Group. Earliest electrocardiographic evidence of myocardial infarction: implications for thrombolytic therapy. BMJ1993;307:409–13.
28. The thrombolysis with a modified dose regimen of recombinant tissue-type plasminogen activator. J Am Coll Cardiol 1989; 14:1566–9.
29. A comparsion of reteplase with alteplase for acute myocardial infarction. The Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO III) Investigators. NEngl J Med 1997;337:1118–23.

10/14/2018