Ankle Brachial Index as a predictor of adverse outcome in patients with acute coronary syndrome undergoing PCI

Submitted for partial fulfillment of master

degree in cardiology

By

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2013

ACKNOWLEDGMENT

My deepest gratitude goes to ALLAH who guided and helped me in this work .

I am deeply indebted to my mentor, **Prof. Dr. Magdy Abdelhamid** who suggested the idea of this work for his patience ,sincere help, guidance and supervision.

I owe too much to **Dr. Hussein Heshmat** for his effort, ideas and guidance throughout the whole work.

I would like also to express my deepest appreciation to all my colleagues for their hearted co-operation and guidance.

Last, but not least, my greatest debt would certainly be to my late father , my mother & my wife for their understanding, patience and devotion without which this work may have never been accomplished.

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September 2013

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LIST OF ABBREVIATIONS

ABI	*Ankle-Brachial Index*
ACC	*American College of Cardiology*
ACE I	*Angiotensin Converting Eenzyme inhibitors*
ACS	*Acute Coronary Syndrome*
ADA	*American Diabetes Association*
AF	*Atrial Fibrillation*
AHA	*American Heart Association*
Apo B	*Apolipoprotein B*
ARTS I	*Arterial Revascularization Therapies Study I*
ATM	*Atmosphere*
BMI	*Body Mass Index*
BMS	*Bare Metal Stents*
BP	*Blood Pressure*
CABG	*Coronary Artery Bypass Grafting*
CAD	*Coronary Artery Disease*
CBVD	*Cerebrovascular Disease*
CCVD	*Cardiac and Cerebrovascular Disease*
CHB	*Complete Heart Block*
CHD	*Coronary Heart Disease*
CHF	*Congestive Heart Failure*
CIN	*Contrast Induced Nephropathy*
CKD	*Chronic Kidney Disease*
CKD-EPI	*Chronic Kidney Disease Epidemiology Collaboration*
CK-MB	*Creatine kinase Myocardial Band Fraction*
CT	*Computed Tomography*
cTn	*Cardiac Troponin*
CVD	*Cardiovascular Disease*
DES	*Drug Eluting Stents*
DM	*Diabetes Mellitus*
DPC	*Drop in Platelet Count*
ECG	*Electrocardiogram*
EF	*Ejection Fraction*
EPIC	*Evaluation of c7E3 for the Prevention of Ischemic Complications*
ESC	*European Society of Cardiology*
EVENT	*Evaluation of Drug Eluting stents and ischemic Events*
FH of CAD	*Family History of Coronary Artery Disease*

GFR	*Glomerular Filtration Rate*
GP	*Glycoprotein*
GPIIb/IIIa	*Glycoprotein II b,IIIa inhibitors*
HDL	*High Density Lipoprotein*
HDL-C	*High Density Lipoprotein –Cholesterol*
HTN	*Hypertension*
ICPS	*Institut Cardiovasculaire Paris Sud*
ICTUS	*Invasive Versus Conservative Treatment in Unstable Coronary Syndromes Investigators*
IVUS	*Intravascular Ultrasound*
JNC	*Joint National Committee*
K/DOQI	*Kidney Disease Outcomes Quality Initiative*
LAD	*Left Anterior Descending artery*
LBBB	*Left Bundle Branch Block*
LCA	*Left Coronary Artery*
LCX	*Left Circumflex Coronary Artery*
LDL-C	*Low Density Lipoprotein –Cholesterol*
LM	*Left Main*
LMWH	*Low Molecular Weight Heparin*
LV	*Left Ventricle*
LVEF	*Left Ventricular Ejection Fraction*
LVH	*Left Ventricular Hypertrophy*
MACE	*Major Adverse Cardiac Events*
MHz	*Mega Hertz*
MI	*Myocardial Infarction*
MVD	*Multivessel Disease*
NKF	*National Kidney Foundation*
NSTEMI	*Non–ST-segment Elevation Myocardial Infarction*
OTT	*Oral Glucose Tolerance Test*
PAD	*Peripheral Arterial Disease*
PAMI	*Primary Angioplasty in Myocardial Infarction*
PCI	*Percutaneous Coronary Intervention*
PMI	*Periprocedural Myocardial Injury*
PTCA	*Percutaneous Transluminal Coronary Angioplasty*
PTFE	*Polytetrafluoroethylene*
PVD	*Peripheral Vascular Disease*
RBBB	*Right Bundle Branch Block*
RCA	*Right Coronary Artery*
SCAI	*Society for Cardiovascular Angiography and Interventions*

SCD	*Sudden Cardiac Death*
SD **STEMI**	*Standard Deviation* *ST segment Elevation Myocardial Infarction*
SVGs	*Saphenous Vein Grafts*
Syntax	*Synergy between Percutaneous Coronary Intervention with Taxus and Cardiac Surgery*
TASC II	*Trans-Atlantic Inter-Society Consensus*
TC	*Total Cholesterol*
TG	*Triglycerides*
TIA	*Transient Ischemic Attack*
TIMI	*Thrombolysis In Myocardial Infarction*
TLR	*Target Lesion Revascularization*
TVR	*Target Vessel Revascularization*
UA/NSTEMI	*Unstable Angina or Non–ST-segment Elevation Myocardial Infarction*
UFH	*Unfractionated heparin*
ULN	*Upper Limit of Normal*
VF	*Ventricular Fibrillation*
VT	*Ventricular Tachycardia*

INTRODUCTION

Atherosclerosis is the most common cause of peripheral arterial disease (PAD) and has the same risk factors as coronary artery disease (CAD).[1] therefore the combination of PAD and CAD is common. PAD has moved from a disease of the lower limbs that is important for local vascular events, such as amputation, to a realization that PAD is a systemic disease showing a very high risk of mortality.

Patients undergoing percutaneous coronary revascularization present an opportunity for physicians to focus on risk factor identification and treatment. Although strides have been made in the treatment of some risk factors such as hyperlipidemia, surprisingly, peripheral arterial disease (PAD) continues to be ignored as a risk factor in cardiology.[2]

To diagnose PAD, guidelines recommend simply measuring the Ankle-Brachial Index (ABI), which is a fast yet effective method of documenting circulatory function in the lower limb. A normal ABI >1.0 indicates good blood flow. However, ABI <0.9 is cause for concern and suggests the presence of PAD, while an ABI <0.5 indicates severe reduction of blood flow. Compared to angiography, an ABI <0.9 is 90% sensitive and 98% specific for a stenosis of 50% or more in arteries of lower limb.[3,4]

PAD is diagnosed (by non-invasive evaluation) in more than 20% of patients undergoing coronary artery angiography and in a third of patients with severe CAD.PAD detected with noninvasive tests is 3-4 times more frequent than intermittent claudication. Based on ABI, PAD is present in less than 5% of individuals younger than 50 years and in more than 20 % of patients older than 70 years old.

Many studies provide data on the effect of intracoronary stent implantation in a high-risk group of patients with combined coronary artery disease and PAD. Peripheral arterial disease is a significant risk factor for lower procedural success and higher in-hospital complications. On follow-up, adverse cardiovascular outcomes were higher in patients with PAD.[5]

Peripheral arterial disease is recognized as an independent predictor of in-hospital complications, including death, stroke, MI and urgent CABG, recurrent ischemia, pulmonary edema, renal failure. Also, PAD is considered a marker for vascular access complications; blood loss requiring transfusion and hematoma.

The likely reasons for such high risk in these patients are adverse baseline and angiographic characteristics, including older age, diabetes mellitus, CHF, lower ejection fraction, renal disease, higher prevalence of multivessel disease, and other characteristics associated with worse in-hospital outcome related to PCI.[6]

Despite the fact that PAD poses a specific hazard to patients undergoing PCI, it is encouraging that some studies found that results of PCI have improved in recent years. This is due in large part to routine coronary stenting and superior generations of stents that are easier to deploy. Advances in pharmacotherapy such as intravenous glycoprotein IIb/IIIa inhibition and clopidogrel also have contributed to this improvement. Ongoing advances such as the incorporation of drug-eluting stents into routine practice will likely further improve the results obtained with PCI in patients with PAD, although this is unlikely to eliminate the gap between patients with and without PAD on end points such as death.

AIM OF THE WORK

To assess the impact of ABI as a predictor of adverse outcome (in-hospital and short term outcome) in patients with ACS undergoing PCI.

ATHEROSCLEROTIC CAD AND ACS

Coronary heart disease (CHD) is a worldwide health epidemic. In the United States, for example, it is estimated that 13.7 million Americans have CHD, including more than 7.2 million individuals who already have had a myocardial infarction. In the group of persons older than 30 years of age, 213 per 100,000 individuals have CHD.[7]

Although age-specific events related to CHD have fallen dramatically in the last few decades, the overall prevalence has risen as populations age and patients survive the initial coronary or cardiovascular event. The Centers for Disease Control and Prevention estimates that life expectancy in America might be increased by 7 years if CHD and its complications were eradicated. Worldwide 30 percent of all deaths can be attributed to cardiovascular disease, of which more than half are caused by CHD, and the forecasts for the future estimate a growing number as a consequence of lifestyle changes in developing countries. Globally, of those dying from cardiovascular diseases, 80 percent are in developing countries and not in the Western world.[8]

CHD represents a continuum of disease pathologies and its subsequent risks. CHD has been classified as chronic CHD, acute coronary syndromes, and sudden death. CHD may present clinically in many ways, extending from an asymptomatic finding to unexpected cardiac collapse. Chronic CHD is always secondary to coronary atherosclerosis, leading to mismatch of coronary blood flow and adenosine triphosphate homeostasis (imbalance of supply and demand) and a stable pattern of coronary ischemia. The clinical pattern includes stable angina pectoris and myocardial hibernation.[9]however; here we focus on a more high-risk population, those with acute coronary syndromes.

Acute Coronary Syndromes

Acute coronary syndrome (ACS) is a unifying term representing a common end result, acute myocardial ischemia. Acute ischemia is usually, but not always, caused by atherosclerotic plaque rupture, fissuring, erosion, or a combination with superimposed intracoronary thrombosis, and is associated with an increased risk of cardiac death and myonecrosis.[10]It encompasses acute myocardial infarction (resulting in ST elevation or non-ST elevation) and unstable angina. Recognizing a patient with ACS is important because the diagnosis triggers both triage and management. Those deemed to have an acute coronary syndrome in the emergency department should be triaged immediately to an area with continuous electrocardiographic monitoring and defibrillation capability. An ECG should be obtained and accurately interpreted within 10 minutes. Those patients with suspected ACS should be managed immediately with antiplatelet and anticoagulant therapies and considered for immediate revascularization mechanically or pharmacologically if new ST-elevation is noted.[11]

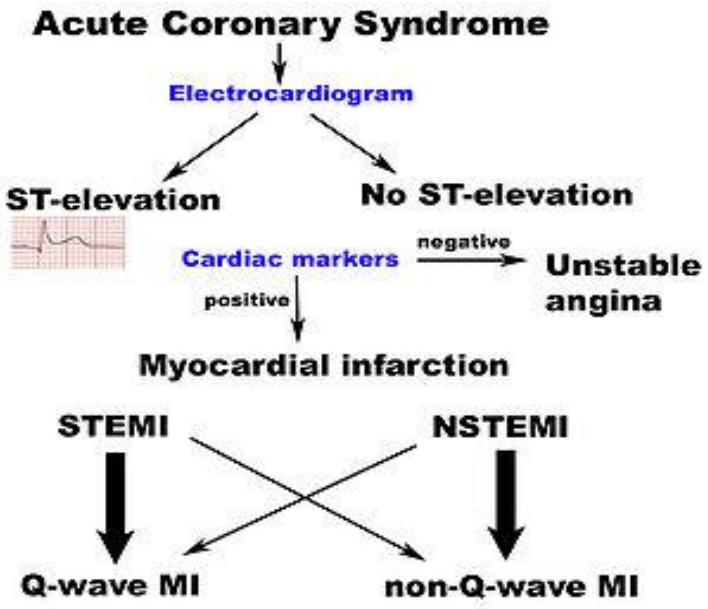

Fig . ACS scheme

Because of the life-threatening nature of an ACS, it is likely to have a low threshold in suspecting a patient with acute chest pain as potentially having an ACS. Because the efficient diagnosis and optimal management of these patients are derived from information mostly only readily available from initial clinical presentation, there is overlap of those with true ACS and those that ultimately do not have CHD as a cause of their cardiac symptoms. In addition, it may not be possible to differentiate patients with myocardial infarction (either ST-elevation or non–ST-elevation) from those with unstable angina in the initial hours as the biomarkers of myonecrosis can be normal initially.[12].

Nonetheless, proper initial triage of patients suspected to have acute coronary ischemia should eventually identify patients as having (1) ACS; (2) a non-ACS cardiovascular condition such as myocarditis/myopericarditis, stress-related cardiomyopathy, aortic dissection, or pulmonary embolism; (3) a non-cardiac cause of chest pain such as gastroesophageal reflux; and (4) a non-cardiac condition that is yet undefined, such as sepsis.[13]

TABLE -1 -- Common Causes of Acute Chest Pain

System	Syndrome	Clinical Description	Key Distinguishing Features
Cardiac	Angina	Retrosternal chest pressure, burning, or heaviness; radiating occasionally to neck, jaw, epigastrium, shoulders, or left arm	Precipitated by exercise, cold weather, or emotional stress; duration <2-10 minutes.
	Rest or unstable angina	Same as angina, but may be more severe	Typically <20 minutes; lower tolerance for exertion
	Acute myocardial infarction	Same as angina, but may be more severe	Sudden onset, usually lasting 30 minutes or longer. Often associated with shortness of breath, weakness, nausea, vomiting
	Pericarditis	Sharp, pleuritic pain aggravated by	Pericardial friction rub

		changes in position; highly variable duration	
Vascular	Aortic dissection	Excruciating, ripping pain of sudden onset in anterior of chest, often radiating to back	Marked severity of unrelenting pain; usually occurs in setting of hypertension or underlying connective tissue disorder such as Marfan syndrome
	Pulmonary embolism	Sudden onset of dyspnea and pain, usually pleuritic with pulmonary infarction	Dyspnea, tachypnea, tachycardia, and signs of right heart failure
	Pulmonary hypertension	Substernal chest pressure, exacerbated by exertion	Pain associated with dyspnea and signs of pulmonary hypertension
Pulmonary	Pleuritis and/or pneumonia	Pleuritic pain, usually brief, over involved area	Pain pleuritic and lateral to midline, associated with dyspnea
	Tracheobronchitis	Burning discomfort in midline	Midline location, associated with coughing
	Spontaneous pneumothorax	Sudden onset of unilateral pleuritic pain, with dyspnea	Abrupt onset of dyspnea and pain
Gastrointestinal	Esophageal reflux	Burning substernal and epigastric discomfort, 10-60 minutes in duration	Aggravated by large meal and postprandial recumbency; relieved by antacid
	Peptic ulcer	Prolonged epigastric or substernal burning	Relieved by antacid or food
	Gallbladder disease	Prolonged epigastric right upper quadrant pain	Unprovoked or following meal
	Pancreatitis	Prolonged, intense epigastric and substernal pain	Risk factors including alcohol, hypertriglyceridemia, and medications
Musculoskeletal	Costochondritis	Sudden onset of intense fleeting pain	May be reproduced by pressure over affected joint; occasional patients have swelling and inflammation over costochondral joint
	Cervical disc disease	Sudden onset of fleeting pain	May be reproduced with movement of neck
	Trauma or strain	Constant pain	Reproduced by palpation or movement of chest wall or arms

Infectious	Herpes zoster	Prolonged burning pain in dermatomal distribution	Vesicular rash, dermatomal distribution
Psychological	Panic disorder	Chest tightness or aching, often accompanied by dyspnea and lasting 30 minutes or more, unrelated to exertion or movement	Patient may have other evidence of emotional disorder

Christopher P.Cannon et al, Braunwald`s heart disease : A textbook of cardiovascular medicine 2007 [14]

ACS patients with new evidence of ST-segment elevation on the presenting ECG are labeled as having an ST-segment elevation myocardial infarction (STEMI) and should be considered for immediate reperfusion therapy by thrombolytics or percutaneous coronary intervention (PCI); those without ST-segment elevation but with evidence of myonecrosis are deemed to have a non–ST-segment elevation myocardial infarction (NSTEMI); and those without any evidence of myonecrosis are diagnosed with unstable angina. [15]

Definition of Unstable Angina

Unstable angina is usually secondary to reduced myocardial perfusion resulting from coronary artery atherothrombosis. In this event, however, the non-occlusive thrombus that developed on a disrupted atherosclerotic plaque does not result in any biochemical evidence of myocardial necrosis. Unstable angina and NSTEMI can be viewed as very closely related clinical conditions with similar presentations and pathogenesis but of differing severity.

Because of the lack of objective data associated with the condition, unstable angina (also known as pre-infarction angina, intermediate coronary syndrome, and