Original Article

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Assessment of Acute Myocardial Infarction by The Use of Special Biochemical Markers

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Background: Acute Myocardial infarction (AMI) occurs during the period when circulation to a region of the heart is obstructed and necrosis ensues. The objective of this study was to estimate the levels of serum Myoglobin and enzymes as cardiac marker for diagnosis of early Myocardial Infarction.

Methodology: This was case control hospital based study involving 130 patients admitted to Elshaab Teaching Hospital and Sudan Heart Center, suffering chest pain, wherein all patients were taken immediately to Coronary Care Unit (CCU). 30 apparently healthy people were enrolled as control. This study was conducted between March and October 2010. The age ranged between (*39-60 years*). Blood samples were collected for Myoglobin using fully automatic immunoanalyzer and cardiac enzymes were measured using Spectrophotometer.

Results: The mean levels of myoglobin concentration in different patients at admission and after 24 hrs of admission to the hospital was 563 ng/ml and 34 ng/ml for patients and control respectively. The level remained higher even after 24 hrs of admission (414 ng/ml). Creatine kinase dramatically elevated within the first 24 hrs in all patients admitted to hospital during 6, 10, 20, 24 and 48 hrs following chest pain attack. CK-MB gradually elevated in patients admitted 6, 10, 20, 24 and 48 hrs from chest pain attack, reaching a peak after 24 hrs of chest pain attack, and then CK-MB levels declined to normal values after 48 hrs. The LDH level exhibited increase levels in patients admitted 6, 10, 20, 24 and 48 hrs from chest pain attack, and the levels persisted all through 24 hrs irrespective of the time admission to hospital following chest pain. AST level was not elevated after 6 hrs or 10 hrs following chest pain attack, but a dramatic elevation was seen after 24 hrs following hospital admission.

Conclusion: This study indicated that Myoglobin and CK-MB are more sensitive cardiac markers compared to total CK, LDH and AST for the diagnosis of Myocardial Infarction.

Keywords: Acute myocardial infarction, myoglobin, creatine kinase, lactate dehydrogenase, aspartate transaminase

Introduction

The heart is a muscular organ responsible for moving blood through a series of vessels to all parts of the body (1). Acute Myocardial Infarction (AMI) occurs during the period when circulation to a region of the heart is obstructed and necrosis ensues (2). AMI is characterized by severe pain (angina pectoris), frequently associated with pallor, perspiration, nausea, shortness of breath, and dizziness. A precursor state of AMI is myocardial ischemia, in which obstruction of a coronary artery leads to severe oxygen deprivation of the myocardium prior to necrosis. Angina may be pronounced during myocardial ischemia (3, 4).

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Studies suggest that between 40 and 50% of nonfatal AMI are unrecognized by patient and are discovered only on subsequent routine ECG or post mortem examination (5). Of this unrecognized infarction, half are truly silent, with the patient unable to recall any symptoms whatsoever (6). The other half of patients with so-called silent infarction can recall an event characterized by symptoms compatible with acute infarction when leading questions are posed after the abnormal ECG is read, unrecognized or silent infarction occurs more commonly in patients without angina, and is more common in patient with diabetes and hypertension.

The diagnosis of AMI, as formally established by the world health organization, requires at least two of the following criteria: a history of chest pain, evolutionary changes on the ECG, and elevation of serial cardiac enzymes (1, 5). Often, the examining physician is fairly certain after obtaining a patients history and completing a physical examination and performing ECG that an MI has occurred. When the ECG fails to demonstrate an AMI, the cardiac markers must be used (6).

Various cardiac markers have been proposed to date some of them are Creatine Kinase (CK); Lactate dehydrogenase (LD); Cardiac Troponin-I and Troponin-T; Myoglobin; Cholesterol; Triglycerides; Low density lipoprotein (LDL); High-density lipoprotein HDL (7). An initial creatine kinase isoenzyme-2 (CK-2) rise takes 4 to 6 h to increase above the upper reference limit. Peak levels occur at approximately 24h. Return to normal takes 48 to 72h. Factors that might affect the classic pattern include size of infarction, CK-2 composition in the myocardium, concomitant skeletal muscle injury, and reperfusion (spontaneous; following thrombolytics, or following angioplasty) (8, 9).

For patients having an Acute Myocardial Infarction (AMI), serum total Lactate Dehydrogenase (LD) values become elevated at 12h to 18h after onset of symptoms, peak at 48h to 72h, and return to below the upper reference limit after 6 to 10 days. LD-1 (The isoenzyme enriched in the heart) rises within 10h to 12h, peaks at 72h to 144h, and returns to normal in approximate 10 days after AMI, paralleling total LD. The elevation patterns of LD-1 and total LD contrast with the elevation patterns of total CK and CK-2, which peak at 24h, and return to below the upper reference limit with 72h after the onset of AMI (10-14). Because of its prolonged half-life, LD-1 is a clinically sensitive (90%) marker for infarction when used more than 24h after occurrence (2, 17).

Studies have shown that myoglobin is a very sensitive marker (90-100%) for AMI. Serum concentrations of myoglobin rise above the reference interval as early as 1 hr after MI, with peak activity in the range of 4 to 12 h, suggesting that serum myoglobin reflects the early course of myocardial necrosis (10, 15).

Myoglobin is rapidly cleared and thus has a substantially reduced clinical sensitivity after 12 h. If myoglobin is to have a role in detecting AMI, it must be within the first 0-4 h, the time period in which CK-2 (CK-MB) is still within its reference interval (16, 22). Also, the overall cumulative release of myoglobin has been correlated with infarct size. However, despite these findings, the measurement of serum myoglobin has not been extensively utilized in clinical laboratories for the routine analysis in AMI. The main reason has been the poor clinical specificity (60-95%) of the protein, caused by the large quantities of myoglobin found in skeletal muscle (3, 11, 14). Other studies that have compared patterns of myoglobin to CK-3 isoforms and total CK activity release have confirmed that myoglobin can be used very early (within 90 min of thrombolytic therapy) for non invasive detection of reperfusion, withhigh specificity (>80%) (15).

Finally, it has been shown that a myoglobin to total CK activity ratio of greater than 5.0 is indicative of reperfusion, with a clinical sensitivity of 75% and a clinical specificity of 96% (19). Thus, it has been concluded that reperfusion status might be satisfactorily predicted by a single sample obtained either at the time of admission, to assess spontaneous reperfusion, or very early (*90 min*) after initiation of thrombolytic therapy (12, 18).

This study was undertaken with the main objective of estimation of serum Myoglobin as cardiac marker for diagnosis of myocardial infarction and compare it with conventional cardiac parameters for early detection of myocytic damage.

Study Design

This study involved 130 patients admitted to Elshaab Teaching Hospital and Sudan Heart Center suffering chest pain, wherein all patients were taken immediately to Coronary Care Unit (CCU) department at the moment of arrival. 30 apparently healthy subjects also participated in this study to serve as control (the control group without present or past history of myocardial infarction).

Blood samples (4 ml) were collected immediately after hospital admission from each patients as well as control using disposable syringes for estimation of cardiac enzymes and for Myoglobin. All blood samples were allowed to clot at room temperature and then centrifuged at 4000 RPM to obtain the serum. The clear serum was taken immediately for analysis or stored at 2 - 8 C^0 for 24 hrs for further use.

Full automatic immunoanalyzer MAGIA 7000 was used for the measurement of Myoglobin. Manual spectrophotometer was used for analysis of the cardiac enzymes. All kits and reagents were procured from sigma chemical company.

Estimation of Myoglobin

Serum is incubated simultaneously with the following reagents: Magnetic particles coated with a monoclonal anti-Mgb antibody. A second monoclonal anti-Mgb antibody, which differs from the first and which is conjugated with alkaline phosphatase (AP conjugate).

After formation of a complex of particles / Mgb / AP conjugate excess conjugate is removed by washing steps. The remaining enzyme activity bound to the particles is directly proportional to the amount of Mgb in the sample (25).

Estimation of Lactate dehydrogenase

Lactate dehydrogenase (LD or LDH) catalyzes the reduction of pyruvate by NADH to form lactate and NAD+. The catalytic concentration is determined from the rate of decrease of NADH measured at 340 nm. The standard methods used for measuring LDH are kinetic methods (1, 12).

Estimation of Creatine Kinase

Creatine Kinase (CK) catalyzes the phosphorylation of ADP, in the presence of creatine phosphate, to form ATP and creatine. The catalytic concentration is determined from the rate of NADPH formation, measured at 340 nm, by means of the hexokinase (HK) and glucose-6-phosphate dehydrogenase (G6P-DH) coupled reactions (13).

Estimation of Creatine Kinase-MB

A specific antibody inhibits CK-M subunits but it does not affect to the CK-B subunits. CK-B catalytic concentration, which corresponds to half of CK-MB concentration, is determined from the rate of NADPH formation, measured at 340 nm, by means of the hexokinase (HK) and glucose-6- phosphate dehydrogenase (G6P-DH) coupled reaction (3, 13).

Table-1: Showing mean values of various biochemical parameters at the time of admission to the hospital and after 24 hours of admission

Parameters		Myoglobin (Mgb)		Creatine Kinase (CK)		Creatine Kinase – MB (CK-MB)		Lactate Dehydrogenase (LDH)		Aspartate Transaminase (AST/GOT)	
Normal Value		*< 72 ng/ml*		*25 - 195 U/L*		*up to 24 U/L*		*235 – 417 U/L*		*< 42 U/L*	
Time of attack	Num. of patients	0 hr	24 hrs	0 hr	24 hrs	0 hr	24 hrs	0 hr	24 hrs	0 hr	24 hrs
Before 6 hr of attack	6	563 ± 163.5	414 ± 113.6	142 ± 46.06	696 ± 223.2	21 ± 2.9	142 ± 58.8	413 ± 117.8	1038 ± 765.5	27 ± 9.9	82 ± 28.7
7– 10 hr	10	468 ± 1273	315 ± 176.8	312 ± 119.7	964 ± 364.3	99 ± 49.9	108 ± 52.07	400 ± 197.04	980 ± 659.6	45 ± 13.4	94 ± 28.7
11 – 20 hr	7	431 ± 147.6	263 ± 121.5	628 ± 239.3	906 ± 427.2	142 ± 64.7	76 ± 25.8	604 ± 186.7	1420 ± 572.2	88 ± 29.8	198 ± 22.6
21 – 24 hr	16	396 ± 177.8	216 ± 177.5	1333 ± 463.5	895 ± 249.2	235 ± 104.09	78 ± 23.3	1454 ± 828.3	2109 ± 944.9	289 ± 138.8	266 ± 105.0
25 – 48 hr	30	230 ± 184.8	113 ± 140.9	725 ± 579.07	444 ± 417.7	141 ± 100.8	39 ± 23.08	1142 ± 701.1	1470 ± 789.3	137 ± 65.3	135 ± 53.3
After 48 hrs	3	86 ± 16.01	64 ± 11.01	359 ± 343.3	106 ± 189.1	18 ± 17.2	13 ± 6.9	1111 ± 66.3	1261 ± 668.6	52 ± 5.1	91 ± 16.5
Normal (no attack)	28	37 ± 10.1	36 ± 9.9	72 ± 24.14	70 ± 22.8	12 ± 2.7	11 ± 2.8	378 ± 53.3	458 ± 49.6	25 ± 7.6	24 ± 8.2
Control	30	34 ± 5.9	35 ± 5.6	54 ± 14.01	55 ± 13.9	10 ± 2.6	10 ± 2.2	283 ± 31.6	285 ± 32.2	20 ± 2.5	20 ± 2.8

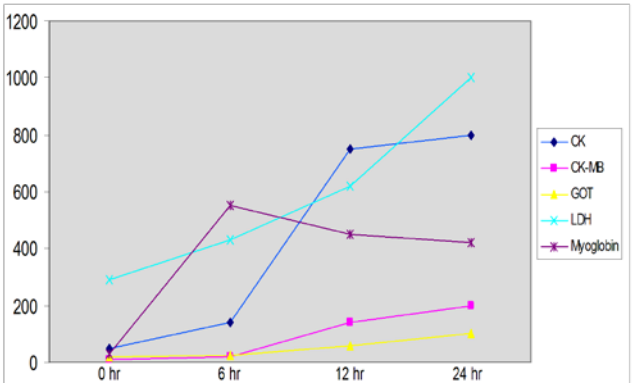

Figure-1: Showing the pattern of myoglobin and activity of marker enzymes from 6 hours of Infarction and up to 24 hours after admission

Estimation Aspartate aminotransferase

Aspartate aminotransferase (AST/GOT) catalyzes the transfer of the amino group from aspartate to 2-oxoglutrate, forming oxalacetate and glutamate. The catalytic concentration is determined from the rate of decrease of NADH measured at 340 nm, by means of the malatedehydrogenase (MDH) coupled reaction (12, 13).

Results

The levels of myoglobin, lactate dehydrogenase, creatine kinase, creatine kinase-MB and apartate transaminase, as the biomarkers for myocardial infarction during 6, 10, 20, 24 and 48 hrs following commencement of chest pain are presented in table No. 1 and the same have been outlined in figure No. 1.

It can be seen that within 6 hrs of onset of chest pain the level of myoglobin was significantly elevated to about 8 fold of normal value and about 16 fold of control value, being 563 ng/ml and 34 ng/ml for patients and control respectively. This level remained higher even after 24 hrs of admission (414 ng/ml).

The same trend was also noticed in other groups of patients admitted hospital at 10, 20, 24 hrs following onset of chest pain. The myoglobin level was much reduced in patients admitted to the hospital after 2 days of the inception of chest pain, but was still significantly higher than normal value or control, and continued to be elevated even after 24 hrs of admission. Patients admitted to the hospital after 3 days of commencement of chest pain, have shown considerable low levels of myoglobin. Concentration reduced to about normal value 24 hrs after that.

Creatine kinase dramatically elevated within the first 24 hrs in all patients admitted to hospital during 6, 10, 20, 24 and 48 hrs following commencement of chest pain. The CK began to fall towards normal values after 24 hrs in patients admitted after 48 hrs of chest pain.

CK-MB gradually elevated in patients admitted 6, 10, 20, 24 and 48 hrs from chest pain attack, reaching a peak after 24 hrs of chest pain attack, then CK-MB levels declined to normal values after 48 hrs.

The LDH level exhibited increase levels in patients admitted 6, 10, 20, 24 and 48 hrs following chest pain attack, and the levels persisted all through 24 hrs irrespective of the time of hospital admission.

AST level was not elevated after 6 hrs or 10 hrs following the chest pain attack, but a dramatic elevation was seen after 24 hrs following hospital admission, however increased levels of AST was noticed in patients admitted to hospital after 24 or 48 hrs following the attack. The levels were within the normal value in patients admitted after 48 hrs of the chest pain attack, and remained so following 24 hrs of admission.

It is worth mentioning here that the levels of Myoglobin, CK, CK-MB, LDH and AST were within the normal range in about 28 % of the patients admitted hospital following chest pain attack.

Discussion

The preliminarily findings obtained from this study revealed that clinical assessment and ECG examinations showed that 72 % of the admitted patients were diagnosed as having myocardial infarction (MI) while 28 % were found to suffer from chest pain.

Cardiac markers were estimated as soon as the patients suffering from chest pain attack were admitted to CCU of Elshab Hospital and Sudan Heart Center. it appeared that myoglobin concentration was immediately elevated during the first six hours of admission, and remain to be so until 24 hours, this pattern of elevation occurred in all patients with chest pain admitted to CCU at 6 hours, 10 hours, 20 hours and 24 hours of chest pain onset, but patients admitted to CCU after 2 days of the onset of chest pain have shown lower value of myoglobin. In all patients, the level of myoglobin decreased to normal value, these findings are in agreement with the results of Mair, et al (20).

Myoglobin and CK-MB are more sensitive in the first hours of attack and this is in agreement with BIOMACS study that reported on patients with chest pain and a non-diagnostic ECG presenting up to 12 hr from the onset of symptoms. Using half-hourly blood samples for

the first 3 hr from admission, it was found that a combination of myoglobin and CK-MB reached a sensitivity of 92% after 2 hr and 98% after 6 hr with the specificity reaching 93% (18).

On the other hand, creatine kinase (CK) and CK-MB gradually elevated in all patients admitted at 6, 10, 20, 24 and 48 hours, and both of them reached peak after 24 hours of chest pain attack. This result is also in line with the work of Ravkilde, et al (22) and Collinson, et al (19)

Lactate dehydrogenase, showed increased levels in patients admitted to CCU at 24 and 48 hours from the onset of the chest pain attack and persisted at high levels till after 72 hours.

Aspartate transferes (AST), also showed elevated values but after 24 hours of chest pain attack, and peak was noticed in patients admitted the hospital after 24 and 48 hours of onset chest pain, but patients admitted to hospital 24 hours after the chest pain have shown normal values of AST. These results are in agreement with the finding of Collinson, et al (19, 23).

Blood samples taken at the moment of hospital admission of patients suffering chest pain attack, and who had a well defined onset of chest pain, showed that during the first 6 hours period, Myoglobin was most significantly sensitive than CK, CK-MB, LDH and GOT. During the 10 – 12 hours of chest pain, the sensitivity of CK and CK-MB were higher than other enzymes, then LDH and GOT sensitivity appear in those came after 24 hr of attack (24).

Our study revealed that, within the 6 – 10 hours after the onset of chest pain, the sensitivity of CK, CK-MB, LDH and GOT (AST) were too low to justify their measurements. The sensitivity of myoglobin was greater after successful early reperfusion. Myoglobin becomes more sensitive during the first 6 hours, while CK, CK-MB and GOT are not sensitive enough during 6 – 10 hours from the onset of chest pain. These findings are in agreement with the results of Mair et al (20), and Bakker et al (4).

As a decrease of myoglobin concentration was seen after 24 hours of chest pain attack, CK and CK-MB activities raised rapidly in patients admitted to hospital 24 hours after the onset of chest pain. An increase of about 879 % and 554 % over the upper reference limit value were seen for CK and CK-MB respectively. These findings are in agreement with the results of William (22) and Rarkidle et al (25) who reported that after myocardial infarction (MI), serum value of CK, was found to increase after about 6 hours, reaches a peak level in 24 – 30 hours and returns to normal in 2 – 4 days.

On the other hand, markers such as LDH and GOT remain unchanged, e.g. within normal values at the first 6 hours of chest pain attack in patients admitted CCU but serum levels raised sharply after 24 hours reaching an increase of about 263 % and 588 % over the upper reference limit for LDH and GOT respectively. These findings agree with the results of Newby, et al (21) who reported that in acute MI serum activity of GOT raised sharply within the first 12 hours, with a peak level at 24 hours or over and return to normal within 3 – 5 days, while LDH activity raised within 12 – 24 hours, reached peak at 48 hours (2 – 4 days) and then return gradually to normal from 8^{th} to 14^{th} day.

Conclusion

This study indicated that Myoglobin and CK-MB are more sensitive cardiac markers compared to total CK, LDH and AST for the diagnosis of Myocardial Infarction. Measuring Myoglobin level could be of great help in confirming the diagnosis of Myocardial Infarction especially in the first few hours following the onset of chest pain.

Recent cardiac marker such as and Myoglobin is very useful in the diagnosis of acute myocardial infarction in the first hour of the attack and more sensitive and specific than cardiac enzyme, recommended to introduce this test in our laboratories. Myoglobin, cardiac Tponin I or T should replace CK and CK-MB during the early period following the onset of chest pain.

Conflict of Interest

The authors declare that no conflict of interest exists in publishing this article.

Reference

1. Abe, J. Yamaguchi, T. Isshiki, T; Myocardial reperfusion can be predicated by myoglobin/creatine kinase ratio of a single blood sample obtained at the time of admission. Am. Heart J., 126: 279-285, 1993.

2. Abramson, J. H; Risk markers for mortality among elderly men community study in Jerusalem, J. Chronic disease; 35: 563-572, 1982.

3. Allain CC, et al; American Heart Association; Coronary Risk Handbook. Estimating Risk of Coronary Heart Disease in daily practice. Clin Chem, 20: 470-475, 1974.

4. Bakker, AJ; Koelemay, MW; Gorgels; J.Failure of new biochemical markers to exclude acute MI at admission Lancer, 342, 1220-2, 1993

5. Chapelle, J.P., Allaf, M.E; Determination of myoglobin in serum by kinetic turbidimetry using the turbitime system. Clin. Chem., 36: 1193, 1990.

6. Drexel, H., Dworzak, E., Kirchmair, W; Myoglobinemia in the early phase of acute myocardial infarction. Am. Heart J., 105: 642-650, 1983.
7. Ellis, A. K., Little, T., Masvil, A. R. Z; Early noninvasive detection of successful reperfusion in patients with acute myocardial infarction. Circulation, 78: 1352-1357, 1988.
8. Gibler, W. B., Lewis, L. M., Erb, R. E; Early detection of acute myocardial infarction in patients presenting with chest pain and ECGs: Serial CK-MB sampling in the emergancy department. Ann. Emerg. Med., 19: 1359-1366, 1990.
9. Gibler, W.B., Gibler, C.D., Weinshenker, E; Myoglobin as an early indicator of acute myocardial infarction. Ann. Emerg. Med., 16: 851-856, 1987.
10. Gibler, W.B., Runyon, J.P., Levy, R.C; A rapid diagnostic and treatment center for patients with chest pain in the emergency department. Ann. Emerg. Med. 25: 1-8,1995.
11. Griesmacher, A., Grimm, M., Schreiner, R., Müller, M.M; Diagnosis of preoperative myocardial infarction by considering relationship of postoperative electrocardiogram changes and enzyme increases after coronary bypass operation. Clin. Chem., 36: 883-887, 1990.
12. IFCC method for the measurement of catalytic concentration of enzyme. J Clin Chem Clin Biochem, 24: 497-510, 1986.
13. IFCC methods for measurement of catalytic concentration of enzyme. JIFCC, 1: 130-139, 1989.
14. Katrukha, A.G., Bereznikova, A.V., Esakova, T.V; Troponin I is released in bloodstream of patients with acute myocardial infarction not in free form but as complex. Clin. Chem., 43: 1379-1385, 1997.
15. Laperche, T., Steg, P.G., Benessiano, J; Patterns of myoglobin and MM creatine kinase isoform release early after intravenous thrombolysis or direct percutaneous transluminal coronary angioplasty for acute myocardial infarction, and implications for the early noninvasive diagnosis of reperfusion. Am. J. Cardiol., 70: 1129-1134, 1992.
16. Lee, T.H., Goldman, L; Serum enzyme assays in diagnosis of acute myocardial infarction. Ann. Intern. Med., 105: 221-233, 1986.
17. Leung, F.T., Galbraith, L.V., Jablonsky, G., Henderson, A.R; Re-evaluation of the diagnostic utility of serum total creatine kinase and creatine kinase-2 in myocardial infarction. Clin Chem., 35: 1435-1440, 1989.
18. Lindahl. B, Venge. P, Wallentin. L; Early diagnosis and exclusion of acute Myocardial Infarction using biochemical monitoring. The BIOMACS study group, biochemical markers of acute coronory syndrome. Coron Artery Dis, 6: 321-8, 1995.
19. Collinson, L. Sylven, C., Santonyi, p; Lactate dehydrogenase and its isoenzyme activities in different parts of normal human heart. Cardiovasc. Res., 23: 601-606, 1989.
20. Mair. S, Morandell. D; Equivalent early sensitivities of Myoglobin, CK-MB, CK isoform ratios and cardiac Troponins I and T for acute MI. Clin. Chem. 41: 1266-72, 1995.
21. Newby, L. K; Gibler, W. B. Ohman, E. M; Biochemical markers in suspected acute MI the need for early assessment. Clin. Chem. 41: 1263-5, 1995.
22. Ravkidle, J., Nissen, H., Horder, M., Thygesen, K; Independent prognostic value of serum creatine kinase isoenzyme MB mass, cardiac troponin T and myosin light chain levels in suspected acute myocardial infarction. J. Am. Coll. Cardiol., 25: 574-581, 1995.
23. Vaananen, H. K., Syrjala, H., Rahkila, P; Serum carbonic anhydrase III and myoglobin concentrations in acute myocardial infarction. Clin. Chem., 36: 635-638, 1990.
24. Wu, A.H.B., Gorent, T.G., Harker, C.C; Role of rapid immunoassay for urgent determinations of creatine kinase isoenzyme MB. Clin. Chem., 35: 1752-1756, 1989.
25. Zabel M, Hohnloser, S.H., Koster, W; Analysis of creatine.kinase, CK-2, myoglobin, and troponin T time-ac0tivity curves for early assessment of coronary artery reperfusion after intravenous thrombolysis. Circulation, 87: 1542-1550, 1993.

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