

RECOMMENDATIONS

1. Measure the enzyme (Lp-PLA₂) mass and genotype detection in parallel to activity measurement in the same study.
2. Further studies on larger number of patients and controls are recommended.

REFERENCES

1. Ross R. Atherosclerosis: an inflammatory disease. *N Engl J Med* 1999; 340:115-26.
2. Jefferson Cardiology Association. What is coronary artery disease?. USA: Jefferson Cardiology Association; 2011.
3. Hansson GK. Mechanisms of disease: inflammation atherosclerosis, and coronary artery disease. *N Engl J Med* 2005; 352(16): 1685–96.
4. Dahlöf B. Cardiovascular disease risk factors: epidemiology and risk assessment. *Am J Cardiol* 2010; 105(1): 3A-9A.
5. Lloyd-Jones DM. Cardiovascular risk prediction: basic concepts, current status, and future directions. *Circulation* 2010; 121(15): 1768–77.
6. Libby P. Inflammation and cardiovascular dis-ease mechanisms. *Am J Clin Nutr* 2006; 83: 456S–60S.
7. Parmar KM, Larman HB, Dai G, Zhang Y, Wang ET, Moorthy SN, et al. Integration of flow-dependent endothelial phenotypes by Kruppel-like factor 2. *J Clin Invest* 2006; 116:49–58.
8. Dai G, Vaughn S, Zhang Y, Wang ET, Garcia-Cardena G, Gimbrone MA Jr. Biomechanical forces in atherosclerosis-resistant vascular regions regulate endothelial redox balance via phosphoinositol 3-kinase/Akt-dependent activation of Nrf2. *Circ Res* 2007; 101:723–33.
9. Libby P, Theroux P. Pathophysiology of coronary artery disease. *Circulation* 2005; 111: 3481–8.
10. Cybulsky MI, Gimbrone MA Jr. Endothelial expression of a mononuclear leukocyte adhesion molecule during atherogenesis. *Science* 1991; 251 (4995):788–91.
11. Kim I, Moon SO, Kim SH, Kim HJ, Koh YS, Koh GY. Vascular endothelial growth factor expression of intercellular adhesion molecule 1 (ICAM-1), vascular cell adhesion molecule 1 (VCAM-1), and E-selectin through nuclear factor-kappa B activation in endothelial cells. *J Biol Chem* 2001;276(10):7614-20
12. Cybulsky MI, Iiyama K, Li H, Zhu S, Chen M, Iiyama M, et al. A major role for VCAM-1, but not ICAM-1, in early atherosclerosis. *J Clin Invest* 2001; 107:1255–62.
13. Jongstra-Bilen J, Haidari M, Zhu SN, Chen M, Guha D, Cybulsky MI. Low-grade chronic inflammation in regions of the normal mouse arterial intima predisposed to atherosclerosis. *J Exp Med* 2006; 203(9):2073–83.
14. Libby P, Ridker PM, Hansson GK. Progress and challenges in translating the biology of atherosclerosis. *Nature* 2011; 473:17–25.
15. Kamei M, Carman CV. New observations on the trafficking and diapedesis of monocytes. *Curr Opin Hematol* 2010(1); 17:43-52.

References

16. Hansson GK, Robertson AK, Söderberg-Nauclér C. Inflammation and atherosclerosis. *Annu Rev Pathol* 2006; 1:297-329.
17. Johnson JL, Newby AC. Macrophage heterogeneity in atherosclerotic plaques. *Curr Opin Lipidol* 2009; 20(5):370-8.
18. Bouhlel MA, Derudas B, Rigamonti E, Dièvert R, Brozek J, Haulon S, et al. PPARgamma activation primes human monocytes into alternative M2 macrophages with anti-inflammatory properties. *Cell Metab* 2007; 6:137-43.
19. Tabas I, Williams KJ, Borén J. Subendothelial lipoprotein retention as the initiating process in atherosclerosis: update and therapeutic implications. *Circulation* 2007; 116(16): 1832-44.
20. Steinberg D. The LDL modification hypothesis of atherogenesis: an update. *J Lipid Res* 2009; 50(Suppl):S376-81.
21. Steinberg D. Atherogenesis in perspective: Hypercholesterolemia and inflammation as partners in crime. *Nat Med* 2002;8(11):1211-7.
22. Chatterjee S, Ghosh N. Oxidized low density lipoprotein stimulates aortic smooth muscle cell proliferation. *Glycobiology* 1996; 6(3):303-11.
23. Maxfield FR, Tabas I. Role of cholesterol and lipid organization in disease. *Nature* 2005; 438 (7068):612-21.
24. Libby P, Ridker PM, Maseri A. The role of inflammation in atherosclerosis. *Circulation* 2002; 105: 1135-42 .
25. Virmani R, Burke AP, Kolodgie FD, Farb A. Vulnerable plaque:the pathology of unstable coronary lesions. *J Interv Cardiol* 2002; 15(6):439-46.
26. Lucas M, Stuart LM, Zhang A, Hodivala-Dilke K, Febbraio M, Silverstein R, et al. Requirements for apoptotic cell contact in regulation of macrophage responses. *J Immunol* 2006;177(6):4047-54.
27. Boyle JJ, Weissberg PL, Bennett MR. Tumor necrosis factor-alpha promotes macrophage induced vascular smooth muscle cell apoptosis by direct and autocrine mechanisms. *Arterioscler Thromb Vasc Biol* 2003; 23(9):1553-8.
28. Newby AC. Metalloproteinase expression in monocytes and macrophages and its relationship to atherosclerotic plaque instability. *Arterioscler Thromb Vasc Biol* 2008;28(12):2108-14.
29. Liu J, Sukhova GK, Sun JS, Xu WH, Libby P, Shi GP. Lysosomal cysteine proteases in atherosclerosis. *Arterioscler Thromb Vasc Biol* 2004; 24:1359-66.
30. West AP, Brodsky IE, Rahner C, Woo DK, Erdjument-Bromage H, Tempst P, et al. TLR signaling augments macrophage bactericidal activity through mitochondrial ROS. *Nature* 2011; 472(7344): 476–80.

31. Miller YI, Viriyakosol S, Binder CJ, Feramisco JR, Kirkland TN, Witztum JL. Minimally modified LDL binds to CD14, induces macrophage spreading via TLR4/MD-2, and inhibits phagocytosis of apoptotic cells. *Journal of Biological Chemistry* 2003; 278(3): 1561–8.
32. Moore KJ, Freeman MW. Scavenger receptors in atherosclerosis: beyond lipid uptake, arteriosclerosis, thrombosis and vascular. *Biology* 2006; 26(8): 1702–11.
33. Lloyd-Jones D, Adams R, Carnethon M, De Simone G, Ferguson B, Flegal K, et al. Heart disease and stroke statistics-- 2009 update. *Circulation* 2009; 119: e21-e181.
34. Howard BV, Rodriguez BL, Bennett PH, Harris MI, Hamman R, Kuller LH, et al. Prevention conference VI: diabetes and cardiovascular disease: Writing Group I: epidemiology. *Circulation* 2002; 105(18):e132-e7.
35. Fuster V, Gotto AM Jr. Risk reduction. *Circulation* 2000; 102 v-94-iv-102.
36. Taddei S, Virdis A, Ghiadoni L, Salvetti G, Bernini G, Magagna A, et al. Age-related reduction of NO availability and oxidative stress in humans. *Hypertension* 2001; 38:274–9.
37. Blackwell KA, Sorenson JP, Richardson DM, Smith LA, Suda O, Nath K, et al. Mechanisms of aging-induced impairment of endothelium-dependent relaxations – role of tetrahydrobiopterin. *Am J Physiol Heart Circ Physiol* 2004; 287: H2448–53.
38. Hawe E, Talmud PJ, Miller GJ, Humphries SE. Family history is a coronary heart disease risk factor in the Second Northwick Park Heart Study. *Annals of Human Genetics* 2003; 67: 97-106.
39. Qureshi N, Armstrong S, Saukko P, Sach T, Middlemass J, Evans PH, et al. Realising the potential of the family history in risk assessment and primary prevention of coronary heart disease in primary care: ADDFAM study protocol. *BMC Health Services Research* 2009; 9:184.
40. Meadows TA, Bhatt DL, Cannon CP, Gersh BJ, Röther J, Goto S, et al. Ethnic differences in cardiovascular risks and mortality in atherothrombotic disease: Insights From the Reduction of Atherothrombosis for Continued Health (REACH) Registry. *Mayo Clin Proc* 2011; 86(10):960-7.
41. Odegaard AO, Koh WP, Gross MD, Yuan JM, Pereira MA. Combined lifestyle factors and cardiovascular disease mortality in Chinese men and women: the Singapore Chinese health study. *Circulation* 2011; 124(25):2847-54.
42. Altman R, Scazziotto A. Role of risk factors in coronary athero-inflammation. *Thrombosis Journal* 2003; 1:1-4.
43. LaRosa JC, Grundy SM, Waters DD, Shear C, Barter P, Fruchart JC, et al. Intensive lipid lowering with atorvastatin in patients with stable coronary disease. *N Engl J Med* 2005; 352(14):1425-35.

References

44. Chobanian AV, Dzau VJ. Renin angiotensin system and atherosclerotic vascular disease. In: Fuster V, Ross R, Topol EJ (eds). *Atherosclerosis and coronary artery disease*. 1sted. Philadelphia: Lippincott-Raven; 2005. 237-40.
45. Boamponsem AG, Boamponsem LK. The role of inflammation in atherosclerosis. *Adv Appl Sci Res* 2011; 2 (4):194-207.
46. Vasan RS, Larson MG, Leip EP, Evans JC, O'Donnell CJ, Kannel WB, et al. Impact of high-normal blood pressure on the risk of cardiovascular disease. *N Engl J Med* 2001; 345(18):1291-7.
47. Katholi RE, Couri DM. Left Ventricular Hypertrophy: Major Risk Factor in Patients with Hypertension: Update and Practical Clinical Applications. *International Journal of Hypertension* 2011;2011: 1-10.
48. Young JL, Libby P (eds). *Atherosclerosis*. In: Lilly LS (ed). *Pathophysiology of heart diseases: a collaborative project of medical student and faculty*. USA: Baltimore: Lippincott Williams & Wilkins; 2007. 123, 132-8.
49. Rea TD, Heckbert SR, Kaplan RC, Smith NL, Lemaitre RN, Psaty BM. Smoking status and risk for recurrent coronary events after myocardial infarction. *Ann Intern Med* 2002; 137(6):494-500.
50. Chi L, Li Y, Stehno-Bittel L, Gao J, Morrison DC, Stehnschulte DJ, Kilepan KN. Interleukin-6 production by endothelial cells via stimulation of protease-activated receptors is amplified by endotoxin and tumor necrosis factor- α . *J Interferon & Cytokine Res* 2001; 21:231-9.
51. Payvar S, Kim S, Rao SV, Krone R, Neely M, Paladugu N, et al. In-Hospital Outcomes of Percutaneous Coronary Interventions in Extremely Obese and Normal-Weight Patients: Findings From the NCDR (National Cardiovascular Data Registry). *J Am College Cardiol* 2013;62(8):692-6.
52. Das SR, Alexander KP, Chen AY, Powell-Wiley TM, Diercks DB, Peterson ED, et al. Impact of body weight and extreme obesity on the presentation, treatment, and in-hospital outcomes of 50,149 patients with ST-segment elevation myocardial infarction results from the NCDR (national cardiovascular data registry). *J Am Coll Cardiol* 2011; 58(25):2642-50.
53. Thompson PD, Buchner D, Pina IL, Balady GJ, Williams MA, Marcus BH, et al. Exercise and physical activity in the prevention and treatment of atherosclerotic cardiovascular disease: a statement from the Council on Clinical Cardiology (Subcommittee on Exercise, Rehabilitation, and Prevention). *Circulation* 2003; 107(24):3109-16.
54. Greenland P, Alpert JS, Beller GA, Benjamin EJ, Budoff MJ, Fayad ZA, et al. 2010 ACCF/AHA guideline for assessment of cardiovascular risk in asymptomatic adults: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol* 2010; 56(25):2182-99.

References

55. Chiuve SE, Fung TT, Rexrode KM, Spiegelman D, Manson JE, Stampfer MJ, et al. Adherence to a low-risk, healthy lifestyle and risk of sudden cardiac death among women. *JAMA* 2011; 306(1):62-9.
56. Lakka HM, Laaksonen DE, Lakka TA, Niskanen LK, Kumpusalo E, Tuomilehto J, et al. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. *JAMA* 2002; 288(21):2709-16.
57. Grundy SM, Cleeman JI, Daniels SR, Donato KA, Eckel RH, Franklin BA, et al. Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. *Circulation* 2005; 112(17):2735-52.
58. Rugulies R. Depression as a predictor for coronary heart disease. a review and meta-analysis. *Am J Prev Med* 2002; 23(1):51-61.
59. Ghiadoni L, Donald AE, Cropley M, Mullen MJ, Oakley G, Taylor M, et al. Mental stress induces transient endothelial dysfunction in humans. *Circulation* 2000; 102(20):2473-8.
60. Hjemdahl P. Stress and the metabolic syndrome: an interesting but enigmatic association. *Circulation* 2002; 106(21):2634-6.
61. Yasojima K, Schwab C, McGeer EG, McGeer PL. Generation of C-reactive protein and complement components in atherosclerotic plaques. *American Journal of Pathology* 2001; 158(3):1039-51.
62. Auer J, Berent R, Eber B. C-reactive protein in patients with acute myocardial infarction. *Jpn Heart J* 2002; 43(6): 607-19.
63. Arroyo-Espliguero R, Avanzas P, Cosín-Sales J, Aldama G, Pizzi C, Kaski JC. C-reactive protein elevation and disease activity in patients with coronary artery disease. *Eur Heart J* 2004; 25(5):401-8.
64. Cojocaru M, Cojocaru IM, Silosi I. Lipoprotein-associated phospholipase A₂ as a predictive biomarker of sub-clinical inflammation in cardiovascular diseases. *Mædica* 2010;5(1):51-55.
65. Szmítko PE, Wang C, Weisel RD, Jeffries GA, Todd J, Biomarkers of vascular disease linking inflammation to endothelial activation part II. *Circulation* 2003; 108: 2041-8.
66. Nordestgaard BG, Chapman MJ, Ray K, Borén J, Andreotti F, Watts GF, et al. Lipoprotein(a) as a cardiovascular risk factor:current status. *European Heart Journal* 2010; 31: 2844-53.
67. Steed MM, Tyagi SC. Mechanisms of cardiovascular remodeling in hyperhomocysteinemia. *Antioxid Redox Signal* 2011; 15(7):1927-43.
68. Mizuno T, Sugimoto M, Matsui H, Hamada M, Shida Y, Yoshioka A. Visual evaluation of blood coagulation during mural thrombogenesis under high shear blood flow. *Thromb Res* 2008; 121:855-64.

References

69. Croce K, Libby P. Intertwining of thrombosis and inflammation in atherosclerosis. *Curr Opin Hematol* 2007; 14:55-61.
70. Otvos JD, Jeyarajah EJ, Cromwell WC. Measurement issues related to lipoprotein heterogeneity. *Am J Cardiol* 2002; 90(8A):22i-9i.
71. Mendivil CO, Rimm EB, Furtado J, Chiuve SE, Sacks FM. Low-Density Lipoproteins containing apolipoprotein c-iii and the risk of coronary heart disease. *Circulation* 2011; 124(19):2065-72.
72. Ridker PM, Libby P. Risk marker for atherothrombotic disease. Bonow RO, Mann DL, Zipes DP, Libby P (eds). *Braunwald's heart disease: a textbook of cardiovascular medicine, single volume: expert consult premium edition - enhanced online features*. 9thed. Philadelphia: Saunders; 2011. 914-34.
73. Go AS, Chertow GM, Fan D, McCulloch CE, Hsu CY. Chronic kidney disease and the risks of death, cardiovascular events, and hospitalization. *N Engl J Med* 2004; 351(13):1296-305.
74. Levy L, Fautrel B, Barnetche T, Schaeffer T. Incidence and risk of fatal myocardial infarction and stroke events in rheumatoid arthritis patients: a systematic review of the literature. *Clin Exp Rheumatol* 2008; 26: 673–9.
75. Chung CP, Oeser A, Raggi P, Gebretsadik T, Shintani AK, Sokka T, et al. Increased coronary-artery atherosclerosis in rheumatoid arthritis: relationship to disease duration and cardiovascular risk factors. *Arthritis Rheum* 2005; 52(10):3045-53.
76. Sani MU. Myocardial disease in human immunodeficiency virus (HIV) infection: a review. *Wien Klin Wochenschr* 2008; 120(3-4):77-87.
77. Malkin CJ, Pugh PJ, Morris PD, Asif S, Jones TH, Channer KS. Low serum testosterone and increased mortality in men with coronary heart disease. *Heart* 2010; 96(22):1821-5.
78. Laugsand LE, Vatten LJ, Platou C, Janszky I. Insomnia and the risk of acute myocardial infarction: a population study. *Circulation* 2011; 124(19):2073-81.
79. Kestenbaum B, Katz R, de Boer I, Hoofnagle A, Sarnak MJ, Shlipak MG, et al. Vitamin d, parathyroid hormone, and cardiovascular events among older adults. *J Am Coll Cardiol* 2011; 58(14):1433-41.
80. 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 Committee for the redefinition of myocardial infarction. *Eur Heart J* 2000; 21:1502–13.
81. Thygesen K, Alpert JS, White HD, Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. Universal definition of myocardial infarction. *Eur Heart J* 2007; 28:2525–38.

References

82. Mendis S, Thygesen K, Kuulasmaa K, Giampaoli S, Mahonen M, Ngu-Blackett K, et al. World Health Organization definition of myocardial infarction: 2008–09 revision. *Int J Epidemiol* 2011; 40:139–46.
83. Solanki R, Solanki S. Relationship of Serum Ferritin with Acute Myocardial Infarction in different age groups. *International Journal of Basic Medicine and Clinical Research* 2014; 1(3):72-5.
84. Bhaskar RT. Preventive cardiology. India: Jaypee Brothers, Medical Publishers; 2011.36.
85. Szmitko PE, Wang C, Weisel RD, De Almeida JR, Anderson TJ, Verma S. New markers of inflammation and endothelial cell activation: Part I. *Circulation* 2003; 108(16):1917-23.
86. Califf R. Ten years of benefit from a one-hour intervention. *Circulation* 1998; 98: 2649-51.
87. Meischke H, Larsen M, Eisenberg M. Gender differences in reported symptoms for acute myocardial infarction: impact on prehospital delay time interval. *The American Journal of Emergency Medicine* 1998; 16:363-6.
88. DeVon H, Ryan C. Chest pain and associated symptoms of acute coronary syndromes. *Journal of Cardiovascular Nursing* 2005; 20: 232-8.
89. Goldberg R, O'Donnell C, Yarzebski J, Bigelow C, Savageau J, Gore J. Sex differences in symptom presentation associated with acute myocardial infarction: a population-based perspective. *American Heart Journal* 1998; 136: 189-95.
90. DeVon H, Ryan C, Ochs A, Shapiro M. Symptoms across the continuum of acute coronary syndromes: differences between women and men. *American Journal of Critical Care* 2008; 17:14-24.
91. King K, McGuire M. Symptom presentation and time to seek care in women and men with acute myocardial infarction. *Heart & Lung* 2007; 36(4):235-43.
92. Omran S, Al Hassan M. Gender differences in signs and symptoms presentation and treatment of Jordanian myocardial infarction patients. *International Journal Of Nursing Practice* 2006; 12:198-204.
93. Arslanian-Engoren C, Patel A, Fang J, Armstrong D, Kline-Rogers E, Duvernoy CS, et al. Symptoms of men and women presenting with acute coronary syndromes. *The American Journal Of Cardiology* 2006; 98:1177-81.
94. Fox K. Acute coronary syndromes: presentation-clinical spectrum and management. *Heart* 2000; 84:93-100.
95. Biomarkers Definitions Working Group. Biomarkers and surrogate endpoints: preferred definitions and conceptual framework. *Clin Pharmacol Ther* 2001; 69:89–95.

References

96. Kemp M, Donovan J, Higham H, Hooper J. Biochemical markers of myocardial injury. *Br J Anaesth* 2004; 93(1):63–73.
97. Alpert JS, Thygesen K, Antman E, Bassand JP. Myocardial infarction redefined--a consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. *Journal of the American College of Cardiology* 2000; 36(3):959-69.
98. Dahadal WA, Dahadal SY. The heart: Laboratory tests of and diagnostic procedure. In: Lee M(ed). *Basic skills in interpreting laboratory data*. USA: American Society of Health-System Pharmacists; 2009. 207-34.
99. Wu AH, Apple FS, Gibler WB, , Jesse RL, Warshaw MM, Valdes R Jr. National academy of clinical biochemistry standards of: laboratory practice recommendations for the use of cardiac markers in coronary artery diseases. *Clin Chem* 1999; 45:1104-21.
100. Bertinchant JP, Larue C, Pernel I, Ledermann B, Fabbro-Peray P, Beck L, et al. Release kinetics of serum cardiac troponin I in ischaemic myocardial injury. *Clin Biochem*. 1996; 29:587-94.
101. Wu AH. A comparison of cardiac troponin T and cardiac troponin I in patients with acute coronary syndromes. *Coron Artery Dis* 1999; 10:69-74.
102. Newby K, Christenson R, Ohman M, Armstrong PW, Thompson TD, Lee KL, et al. Value of serial troponin T measures for early and late risk stratification in patients with acute coronary syndromes. *Circulation* 1998; 98:1853-9.
103. Ohman EM, Armstrong PW, Christenson RH, et al. For the GUSTO IIA Investigators. Cardiac troponin T levels for risk stratification in acute myocardial ischemia. *N Engl J Med* 1996; 335:1333-41.
104. Antman EM, Tanasijevic MJ, Thompson B, Schactman M, McCabe CH, Cannon CP, et al. Cardiac-specific troponin I levels to predict the risk of mortality in the patients with acute coronary syndromes. *N Engl J Med* 1996; 335(18): 1342-9.
105. Morrow DA, Cannon CP, Jesse RL, Newby LK, Ravkilde J, Storrow AB, et al. National academy of clinical biochemistry laboratory medicine practice guidelines: clinical characteristics and utilization of biochemical markers in acute coronary syndromes. *Circulation*. 2007; 115: e356-75.
106. Anderson JL, Adams CD, Antman EM, Bridges CR, Califf RM, Casey DE Jr, Chavey WE 2nd, et al. ACC/AHA 2007 guidelines for the management of patients with unstable angina/non-ST-Elevation myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non ST -Elevation Myocardial Infarction) developed in collaboration with the American College of Emergency Physicians, the Society for Cardiovascular Angiography and Interventions and the Society of Thoracic Surgeons endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation and the Society for Academic Emergency Medicine. *J Am Coll Cardiol* 2007; 50(7):e1-e157.

References

107. Bruns CA, Ashwood ER, Bruns DE. Tietz textbook of clinical chemistry and molecular diagnosis. 5thed. St. Louis, USA: Elsevier; 2012.
108. O'Neill S, Snowden CP. Monitoring in the vascular surgery patient. In: Moores C, Nimmo AF (eds). Core topics in vascular anaesthesia. UK: Cambridge University Press; 2012. 52-61.
109. Schlattner U, Tokarska-Schlattner M, Wallimann T. Mitochondrial creatine kinase in human health and disease. *Biochimica et Biophysica Acta—Molecular Basis of Disease* 2006; 1762:164-80.
110. Saenger AK, Jaffe AS. Requiem for a heavyweight: the demise of creatine kinase-MB. *Circulation* 2008;118(21):2200-6.
111. Jaffe AS, Babuin L, Apple FS. Biomarkers in acute cardiac disease. *J Am Coll Cardiol* 2006; 48: 1-11.
112. Vaidya HC. Myoglobin: an early biochemical marker for the diagnosis of acute myocardial infarction. *J Clin Immunoassay* 1994;17:35-9.
113. Lawrence LW, Broussard LA. Prophyryns and hemoglobin. In: Bishop ML, Fody EP, Schoeff LE (eds). *Clinical chemistry: principles, techniques, and correlations*. Philadelphia: Lippincott Williams & Wilkins; 2013. 415-36.
114. Dasgupta A, Wahed A. *Clinical chemistry, immunology and laboratory quality control: a comprehensive review for board preparation, certification and clinical practice*. Philadelphia: Elsevier Science; 2013.
115. Pepys MB, Berger A. The renaissance of C reactive protein. *BMJ* 2001; 4:322-5.
116. Labarrere CA, Zaloga GP. C-reactive protein: from innocent bystander to pivotal mediator of atherosclerosis. *Am J Med* 2004; 117:499–507.
117. Black S, Kushner I, Samols D. C-reactive protein. *J Biol Chem* 2004; 48487:279-90.
118. Blake GJ, Ridker PM. C-reactive protein and other inflammatory risk markers in acute coronary syndromes. *J Am Coll Cardiol* 2003; 41:37-42.
119. Azzazy HM, Pelsers MM, Christenson RH. Unbound free fatty acids and heart-type fatty acid-binding protein: diagnostic assays and clinical applications. *Clin Chem* 2006; 52:19–29.
120. Liuzzo B, Colizzi L, Rizzello V. Clinical use of C-reactive protein for the prognostic stratification of patients with ischemic heart disease. *Ital Heart J.* 2001; 2: 164-71.
121. Ridker PM, Rifai N, Cook NR, Bradwin G, Buring JE. Non-HDL cholesterol, apolipoproteins A-I and B100, standard lipid measures, lipid ratios, and CRP as risk factors for cardiovascular disease in women. *J Am Med Assoc* 2005; 294:326–33.

References

122. Calabro P, Willerson JT, Yeh ET. Inflammatory cytokines stimulated C-reactive protein production by human coronary artery smooth muscle cells. *Circulation* 2003; 108:1930–2.
123. Rifai N, Ridker PM. High-sensitivity C-reactive protein: a novel and promising marker of coronary heart disease. *Clin Chem* 2001;47(3):403-11.
124. Brennan ML, Penn MS, Van Lente F, Nambi V, Shishehbor MH, Aviles RJ, et al. Prognostic value of myeloperoxidase in patients with chest pain. *N Engl J Med* 2003; 349:1595–604.
125. Pennathur S, Bergt C, Shao B, Byun J, Kassim SY, Singh P, et al. Human atherosclerotic intima and blood of patients with established coronary artery disease contain high density lipoprotein damaged by reactive nitrogen species. *J Biol Chem* 2004;279(41):42977-83.
126. Sugiyama S, Okada Y, Sukhova GK, Virmani R, Heinecke JW, Libby P. Macrophage myeloperoxidase regulation by granulocyte macrophage colony-stimulating factor in human atherosclerosis and implications in acute coronary syndromes. *Am J Pathol* 2001; 158:879–91.
127. Bonaca MP, Scirica BM, Sabatine MS, Jarolim P, Murphy SA, Chamberlin JS, et al. Prospective evaluation of pregnancy-associated plasma protein-a and outcomes in patients with acute coronary syndromes. *J Am Coll Cardiol* 2012;60(4):332-8.
128. Bayes-Genis A, Conover CA, Overgaard MT, Bailey KR, Christiansen M, Holmes DR Jr, et al. Pregnancy-associated plasma protein A as a marker of acute coronary syndromes. *N Engl J Med* 2001; 345:1022–9.
129. Apple FS, Wu AH, Mair J, Ravkilde J, Panteghini M, Tate J, et al. Future biomarkers for detection of ischemia and risk stratification in acute coronary syndrome. *Clin Chem* 2005; 51:810–24.
130. Heeschen C, Dimmeler S, Hamm CW, van den Brand MJ, Boersma E, Zeiher AM, et al. Soluble CD40 ligand in acute coronary syndromes. *N Engl J Med* 2003; 348:1104–11.
131. Varo N, de Lemos JA, Libby P, Morrow DA, Murphy SA, Nuzzo R, et al. Soluble CD40L: risk prediction after acute coronary syndromes. *Circulation* 2003; 108:1049–52.
132. Tamura N, Kobayashi S, Kato K, Bando H, Haruta K, Oyanagi M, et al. Soluble CD154 in rheumatoid arthritis: elevated plasma levels in cases with vasculitis. *J Rheumatol* 2001; 28:2583–90.
133. Roy D, Quiles J, Aldama G, Sinha M, Avanzas P, Arroyo-Espliguero R, et al. Ischemia modified albumin for the assessment of patients presenting to the emergency department with acute chest pain but normal or non-diagnostic 12-lead electrocardiograms and negative cardiac troponin T. *Int J Cardiol* 2004;97:297–301.

References

134. Wu AHB, Crosby P, Fagan G, Danne O, Frei U. Ischemia-modified albumin, free fatty acids, whole blood choline, B-type natriuretic peptide, glycogen phosphorylase BB, and cardiac troponin. In: Wu AHB (ed). *Cardiac markers*. 2nded. Totowa, New Jersey: Humana Press; 2003. 259-77.
135. Martinez-Rumayor A, Richards M, Burnett JC, Januzzi JL. Biology of the natriuretic peptides. *Am J Cardiol* 2008; 101 (Suppl 1): 3A-8A.
136. Daniels LB, Maisel AS. Natriuretic peptides. *J Am Coll Cardiol*. 2007; 50: 2357-68.
137. Baggish AL, van Kimmenade RR, Januzzi JL. The differential diagnosis of an elevated amino-terminal pro-B-type natriuretic peptide level. *Am J Cardiol* 2008; 101 (3A): 43-8.
138. Naik P. *Essentials of biochemistry*. 1sted. India: Jaypee Brothers; 2011.371.
139. Kudo I, Murakami M. Phospholipase A₂ enzymes. *Prostaglandins Other Lipid Mediat* 2002; 68–9.
140. Schaloske RH, Dennis EA. The phospholipase A₂ superfamily and its group numbering system. *Biochim Biophys Acta* 2006; 1761:1246-59.
141. Pratico D. Prostanoid and isoprostanoid pathways in atherogenesis. *Atherosclerosis* 2008; 201:8–16.
142. Deigner HP, Hermetter A. Oxidized phospholipids: emerging lipid mediators in pathophysiology. *Curr Opin Lipidol* 2008; 19:289–94.
143. Wymann MP, Schneider R. Lipid signalling in disease. *Nat Rev Mol Cell Biol* 2008; 9:162–76.
144. Das UN. Can endogenous lipid molecules serve as predictors and prog-nostic markers of coronary heart disease?. *Lipids Health Dis* 2008; 7:19.
145. Shimizu T. Lipid mediators in health and disease: enzymes and receptors as therapeutic targets for the regulation of immunity and inflammation. *Annu Rev Pharmacol Toxicol* 2009; 49: 123–50.
146. Cedars A, Jenkins CM, Mancuso DJ, Gross RW. Calcium-independent phospholipases in the heart: mediators of cellular signaling, bioenergetics and ischemia-induced electrophysiologic dysfunction. *J Cardiovasc Pharmacol* 2009; 53:277–89.
147. Burke JE, Dennis EA. Phospholipase A₂ biochemistry. *Cardiovasc Drugs Ther* 2009; 49:23-59.
148. Stafforini DM. Biology of platelet-activating factor acetylhydrolase (PAF-AH, lipoprotein associated phospholipase A₂). *Cardiovasc Drugs Ther* 2009; 23:73–83.
149. Lambeau G, Gelb MH. Biochemistry and physiology of mammalian secreted phospholipases A₂. *Annu Rev Biochem* 2008; 77:495–520.

References

150. Ii H, Hontani N, Toshida I, Oka M, Sato T, Akiba S. Group IVA phospholipase A₂-associated production of MMP-9 in macrophages and formation of atherosclerotic lesions. *Biol Pharm Bull* 2008; 31:363–8.
151. Herbert SP, Odell AF, Ponnambalam S, Walker JH. Activation of cytosolic phospholipase A₂-as a novel mechanism regulating endo-thelial cell cycle progression and angiogenesis. *J Biol Chem* 2009; 284:5784-96.
152. White MC, McHowat J. The therapeutic potential of phospholipase A₂ inhibitors in cardiovascular disease. *Cardiovasc Hematol Agents Med Chem* 2007; 5:91–5.
153. Mishra RS, Carnevale KA, Cathcart MK. iPLA₂beta: front and center in human monocyte chemotaxis to MCP-1. *J Exp Med* 2008; 205:347–59.
154. Zalewski A, Macphee C. Role of lipoprotein-associated phospholipase A₂ in atherosclerosis: biology, epidemiology, and possible therapeutic target. *Arterioscler Thromb Vasc Biol* 2005; 25:923–31.
155. Oorni K, Kovanen PT. Lipoprotein modification by secretory phospho-lipase A₂ enzymes contributes to the initiation and progression of atherosclerosis. *Curr Opin Lipidol* 2009; 20:421–7.
156. McIntyre TM, Prescott SM, Stafforini DM. The emerging roles of PAF acetylhydrolase. *J Lipid Res* 2009; 50(suppl):S9–S255.
157. Tselepis AD, John Chapman M. Inflammation, bioactive lipids and atherosclerosis: potential roles of a lipoprotein-associated phospholipase A₂, platelet activating factor-acetylhydrolase. *Atheroscler Suppl* 2002;3:57– 68.
158. Blencowe C, Hermetter A, Kostner GM, Daigner HP. Enhanced association of platelet-activating factor acetylhydrolase with lipoprotein(a) in comparison with low density lipoprotein. *J Biol Chem* 1995; 270:31151-7.
159. Tsimikas S, Brilakis ES, Miller ER, McConnell JP, Lennon RJ, Kornman KS, et al. Oxidized phospholipids, Lp(a) lipoprotein, and coronary artery disease. *N Engl J Med* 2005; 353:46-57.
160. Bossola M, Tazza L, Merki E, Giungi S, Luciani G, Miller ER, et al. Oxidized low-density lipoprotein biomarkers in patients with end-stage renal failure: acute effects of hemodialysis *Blood Purif* 2007; 25:457–65.
161. Tjoelker LW, Eberhardt C, Unger J, Trong HL, Zimmerman GA McIntyre TM, et al. Plasma platelet-activating factor acetylhydrolase is a secreted phospholipase A₂ with a catalytic triad. *J Biol Chem* 1995; 270:25481–7.
162. Samanta U, Bahnson BJ. Crystal structure of human plasma platelet-activating factor acetylhydrolase: structural implication to lipoprotein binding and catalysis. *J Biol Chem* 2008; 283:31617-24.

References

163. Stafforini DM, Sheller JR, Blackwell TS, Sapirstein A, Yull FE, McIntyre TM, et al. Release of free F2-isoprostanes from esterified phospholipids is catalyzed by intra-cellular and plasma platelet-activating factor acetylhydrolases. *J Biol Chem* 2006; 281:4616–23.
164. Kriska T, Marathe GK, Schmidt JC, McIntyre TM, Girotti AW. Phospholipase action of platelet-activating factor acetylhydrolase, but not paraoxonase-1, on long fatty acyl chain phospholipid hydroperoxides. *J Biol Chem* 2007; 282:100–8.
165. Bui QT, Prempeh M, Wilensky RL. Atherosclerotic plaque development. *Int J Biochem Cell Biol* 2009; 41:2109–13.
166. Colley KJ, Wolfert RL, Cobble ME. Lipoprotein associated phospholipase A₂: role in atherosclerosis and utility as a biomarker for cardiovascular risk. *EPMA Journal* 2011; 2: 27–38.
167. Ninio E. Phospholipid mediators in the vessel wall: involvement in atherosclerosis. *Curr Opin Clin Nutr Metab Care* 2005; 8:123–31.
168. Asseman C, Mauze S, Leach MW, Coffman RL, Powrie F. An essential role for interleukin 10 in the function of regulatory T cells that inhibit intestinal inflammation. *J Exp Med* 1999; 190: 995–1004.
169. Bochkov VN, Kadl A, Huber J, Gruber F, Binder BR, Leitinger N. Protective role of phospholipid oxidation products in endotoxin-induced tissue damage. *Nature* 2002; 419:77–81.
170. Tellis CC, Tselepis AD. The role of lipoprotein-associated phospholipase A₂ in atherosclerosis may depend on its lipoprotein carrier in plasma. *Biochim Biophys Acta* 2009; 1791:327–38.
171. Carpenter KL, Dennis IF, Challis IR, Osborn DP, Macphee CH, Leake DS, et al. Inhibition of lipoprotein-associated phospholipase A₂ diminishes the death-inducing effects of oxidised LDL on human monocyte-macrophages. *FEBS Lett* 2001; 505:357-63.
172. Shi Y, Zhang P, Zhang L, Osman H, Mohler ER 3rd, Macphee C, et al. Role of lipoprotein-associated phospholipase A₂ in leukocyte activation and inflammatory responses. *Atherosclerosis* 2007; 191:54-62.
173. Kabarowski JH. G2A and LPC: regulatory functions in immunity. *Prostaglandins Other Lipid Mediat* 2009; 89:73–81.
174. Aprahamian T, Rifkin I, Bonegio R, Hugel B, Freyssinet JM, Sato K, et al. Impaired clearance of apoptotic cells promotes synergy between atherogenesis and autoimmune disease. *J Exp Med* 2004; 199:1121-31.
175. Mallat Z, Lambeau GR, Tedgui A. Lipoprotein-associated and secreted phospholipases A₂ in cardiovascular disease roles as biological effectors and biomarkers. *Circulation* 2010; 122:2183-200.

References

176. Kolodgie FD, Burke AP, Skorija KS, Ladich E, Kutys R, Makuria AT, et al. Lipoprotein-associated phospholipase A₂ protein expression in the natural progression of human coronary atherosclerosis. *Arterioscler Thromb Vasc Biol* 2006; 26:2523–9.
177. Munzel T, Gori T. Lipoprotein-associated phospholipase A₂ a marker of vascular inflammation and systemic vulnerability. *European Heart Journal* 2009; 30: 2829–31.
178. Ballantyne CM, Hoogeveen RC, Bang H, Coresh J, Folsom AR, Chambless LE, et al. Lipoprotein-associated phospholipase A₂, high sensitivity C-reactive protein and risk for incident ischemic stroke in middle-aged men and women in the Atherosclerosis Risk in Communities (ARIC) study. *Arch Intern Med* 2005; 165:2479–84.
179. Sacks DB, Carbohydrates. In: Burtis CA, Ashwood ER, Bruns DE (eds). *Teitz textbook of clinical chemistry and molecular diagnostics*. 4th ed. St Louis: Elsevier Saunders; 2006. 869-72.
180. Panteghini M, Bais R, van Solinge WW. Enzymes. In: Burtis CA, Ashwood ER, Bruns DE (eds). *Teitz textbook of clinical chemistry and molecular diagnostics*. 4th ed. St Louis: Elsevier Saunders; 2006. 602-7.
181. Rifai N, Ballantyne CM, Cushman M, Levy D, Myers GL. Point: high-sensitivity C-reactive protein and cardiac C-reactive protein assays: is there a need to differentiate?. *Clin Chem* 2006;52(7):1254-6.
182. Apple FS, Quist HE, Doyle PJ, Otto AP, Murakami MM. Plasma 99th percentile reference limits for cardiac troponin and creatine kinase MB mass for use with European Society of Cardiology/American College of Cardiology consensus recommendations. *Clin Chem* 2003;49(8):1331-6.
183. Collinson PO, Boa FG, Gaze DC. Measurement of cardiac troponins. *Ann Clin Biochem* 2001;38(Pt 5):423-49.
184. Apple FS, Jaffe AS. Cardiac function. In: Burtis CA, Ashwood ER, Bruns DE (eds). *Teitz textbook of clinical chemistry and molecular diagnostics*. 4th ed. St Louis: Elsevier Saunders; 2006. 1635-40.
185. diaDexus. PLAC® Test for Lp-PLA₂ activity: Enzyme Assay for the Quantitative Determination of Lp-PLA₂ Activity in human serum or plasma. USA: South San Francisco: diaDexus; 2014.
186. Kotz S, Balakrishnan N, Read CB, Vidakovic B. *Encyclopedia of statistical sciences*. 2nd ed. Hoboken, New Jersey: Wiley-Interscience; 2006.
187. Kirkpatrick LA, Feeney BC. *A simple guide to IBM SPSS statistics for version 20.0*. Student ed. Belmont, Calif.: Wadsworth, Cengage Learning; 2013.

References

188. Pearson TA, Bazzarre TL, Daniels SR, Fair JM, Fortmann SP, Franklin BA, et al. American Heart Association Guide for Improving Cardiovascular Health at the Community Level: A Statement for Public Health Practitioners, Healthcare Providers, and Health Policy Makers From the American Heart Association Expert Panel on Population and Prevention Science. *Circulation* 2003;107(4):645-51.
189. Cai A, Zheng D, Qiu R, Mai W, Zhou Y. Lipoprotein-associated phospholipase A₂ (Lp-PLA₂): a novel and promising biomarker for cardiovascular risks assessment. *Dis Markers* 2013;34(5):323-31.
190. Libby P. What have we learned about the biology of atherosclerosis? The role of inflammation. *Am J Cardiol* 2001;88(7b):3j-6.
191. Libby P. Act local, act global: inflammation and the multiplicity of "vulnerable" coronary plaques. *J Am Coll Cardiol* 2005;45(10):1600-2.
192. Hansson GK, Libby P, Schonbeck U, Yan ZQ. Innate and adaptive immunity in the pathogenesis of atherosclerosis. *Circ Res* 2002;91(4):281-91.
193. Wilensky RL, Hamamdzcic D. The molecular basis of vulnerable plaque: potential therapeutic role for immunomodulation. *Curr Opin Cardiol* 2007;22(6):545-51.
194. Dada N, Kim NW, Wolfert RL. Lp-PLA₂: an emerging biomarker of coronary heart disease. *Expert Rev Mol Diagn* 2002;2(1):17-22.
195. Bochkov VN, Oskolkova OV, Birukov KG, Levonen AL, Binder CJ, Stockl J. Generation and biological activities of oxidized phospholipids. *Antioxid Redox Signal* 2010;12(8):1009-59.
196. Dada N, Kim NW, Wolfert RL. Lp-PLA₂: an emerging biomarker of coronary heart disease. *Expert Rev Mol Diagn* 2002;2(1):17-22.
197. Burchardt P, Zurawski J, Zuchowski B, Kubacki T, Murawa D, Wiktorowicz K, et al. Low-density lipoprotein, its susceptibility to oxidation and the role of lipoprotein-associated phospholipase A₂ and carboxyl ester lipase lipases in atherosclerotic plaque formation. *Arch Med Sci* 2013;9(1):151-8.
198. Packard CJ, O'Reilly DS, Caslake MJ, McMahon AD, Ford I, Cooney J, et al. Lipoprotein-associated phospholipase A₂ as an independent predictor of coronary heart disease. West of Scotland Coronary Prevention Study Group. *N Engl J Med* 2000;343(16):1148-55.
199. Corson MA, Jones PH, Davidson MH. Review of the evidence for the clinical utility of lipoprotein-associated phospholipase A₂ as a cardiovascular risk marker. *Am J Cardiol* 2008;101(12a):41f-50f.
200. Ballantyne CM, Hoogeveen RC, Bang H, Coresh J, Folsom AR, Heiss G, et al. Lipoprotein-associated phospholipase A₂, high-sensitivity C-reactive protein, and risk for incident coronary heart disease in middle-aged men and women in the Atherosclerosis Risk in Communities (ARIC) study. *Circulation* 2004;109(7):837-42.

References

201. Lavi S, McConnell JP, Rihal CS, Prasad A, Mathew V, Lerman LO, et al. Local production of lipoprotein-associated phospholipase A₂ and lysophosphatidylcholine in the coronary circulation: association with early coronary atherosclerosis and endothelial dysfunction in humans. *Circulation* 2007;115(21):2715-21.
202. Oei HH, van der Meer IM, Hofman A, Koudstaal PJ, Stijnen T, Breteler MM, et al. Lipoprotein-associated phospholipase A₂ activity is associated with risk of coronary heart disease and ischemic stroke: the Rotterdam Study. *Circulation* 2005;111(5):570-5.
203. Cook NR, Paynter NP, Manson JE, Martin LW, Robinson JG, Wassertheil-Smoller S, et al. Clinical utility of lipoprotein-associated phospholipase A₂ for cardiovascular disease prediction in a multiethnic cohort of women. *Clin Chem* 2012;58(9):1352-63.
204. Winkler K, Hoffmann MM, Winkelmann BR, Friedrich I, Schafer G, Seelhorst U, et al. Lipoprotein-associated phospholipase A₂ predicts 5-year cardiac mortality independently of established risk factors and adds prognostic information in patients with low and medium high-sensitivity C-reactive protein (the Ludwigshafen risk and cardiovascular health study). *Clin Chem* 2007;53(8):1440-7.
205. Rallidis LS, Tellis CC, Lekakis J, Rizos I, Varounis C, Charalampopoulos A, et al. Lipoprotein-associated phospholipase A(2) bound on high-density lipoprotein is associated with lower risk for cardiac death in stable coronary artery disease patients: a 3-year follow-up. *J Am Coll Cardiol* 2012;60(20):2053-60.
206. Jenny NS, Solomon C, Cushman M, Tracy RP, Nelson JJ, Psaty BM, et al. Lipoprotein-associated phospholipase A(2) (Lp-PLA(2)) and risk of cardiovascular disease in older adults: results from the Cardiovascular Health Study. *Atherosclerosis* 2010;209(2):528-32.
207. Tsimikas S, Willeit J, Knoflach M, Mayr M, Egger G, Notdurfter M, et al. Lipoprotein-associated phospholipase A₂ activity, ferritin levels, metabolic syndrome, and 10-year cardiovascular and non-cardiovascular mortality: results from the Bruneck study. *Eur Heart J* 2009;30(1):107-15.
208. Hatoum IJ, Hu FB, Nelson JJ, Rimm EB. Lipoprotein-associated phospholipase A₂ activity and incident coronary heart disease among men and women with type 2 diabetes. *Diabetes* 2010;59(5):1239-43.
209. Hatoum IJ, Cook NR, Nelson JJ, Rexrode KM, Rimm EB. Lipoprotein-associated phospholipase A₂ activity improves risk discrimination of incident coronary heart disease among women. *Am Heart J* 2011;161(3):516-22.
210. Maiolino G, Pedon L, Cesari M, Frigo AC, Wolfert RL, Barisa M, et al. Lipoprotein-associated phospholipase A₂ activity predicts cardiovascular events in high risk coronary artery disease patients. *PLoS One* 2012;7(10):e48171.

References

211. Persson M, Hedblad B, Nelson JJ, Berglund G. Elevated Lp-PLA₂ levels add prognostic information to the metabolic syndrome on incidence of cardiovascular events among middle-aged nondiabetic subjects. *Arterioscler Thromb Vasc Biol* 2007;27(6):1411-6.
212. Anuurad E, Ozturk Z, Enkhmaa B, Pearson TA, Berglund L. Association of lipoprotein-associated phospholipase A₂ with coronary artery disease in African-Americans and Caucasians. *J Clin Endocrinol Metab* 2010;95(5):2376-83.
213. Liu CF, Qin L, Ren JY, Chen H, Wang WM, Liu J, et al. Elevated plasma lipoprotein-associated phospholipase A(2) activity is associated with plaque rupture in patients with coronary artery disease. *Chin Med J (Engl)* 2011;124(16):2469-73.
214. da Silva IT, Timm Ade S, Damasceno NR. Influence of obesity and cardiometabolic makers on lipoprotein-associated phospholipase A₂ (Lp-PLA₂) activity in adolescents: the healthy young cross-sectional study. *Lipids Health Dis* 2013;12:19.

المُلخَص العَرَبِي

مرض الشريان التاجي هو عبارة عن حالة حيث تتكون الويحات داخل الشرايين التاجية المسؤولة عن تروية عضلة القلب بالدم الغني بالأكسجين.

امراض الشرايين التاجية هي اشهر اشكال امراض القلب والاعوية الدموية والسبب الرئيسي لها هو تصلب الشرايين ، تصلب الشرايين هو مرض مزمن للشرايين المتوسطة والكبيرة الحجم والذي يتميز بتكون الويحات

الفسفوليبيز المرتبط بالبروتين الدهنى أ٢ والمعروف ايضا بأسم الاسيتيل هيدروليز لعامل تفعيل الصفائح الدموية هو علامة بيولوجية حديثة تفرز من الخلايا الالتهابية المشاركة في وحة تصلب الشرايين وبالتالي فهو مخصص للغاية لالتهابات الاعوية الدموية وليس للالتهابات النظامية.

الفسفوليبيز المرتبط بالبروتين الدهنى أ٢ له تخصص واسع التأثير على الدهون الفوسفاتية المؤكسده وهذا يوضح التأثير المتخصص للفسفوليبيز المرتبط بالبروتين الدهنى أ٢ فى التخلص من الدهون الفوسفاتية المؤكسده من البروتونات الدهنيه مع توليد الفوسفاتيديل كولين المنحل والاحماض الدهنية الحرة المؤكسده والذان يعتبران وسطاء للالتهابات المسؤولة عن تصلب الشرايين.

مع ذلك، فإن الدراسة الحالية تهدف الى قياس نشاط إنزيم الفوسفوليبيز المرتبط بالبروتين الدهنى أ٢ فى مرضى يعانون من احتشاء عضلة القلب فى الشعب المصرى ، لقد تم إختيار ٣٠ مريضاً مصرياً من قسم امراض القلب فى المستشفى الرئيسى الجامعى بالاسكندرية يشملون ٦٦,٧% ذكور ، ٣٣,٣% اناث والذين تتراوح اعمارهم من ٣٥- ٦١ عاماً بمتوسط قدره ٤٨ عاماً والذين يعانون من احتشاء عضلة القلب وهو ماتأكد بمخطط القلب الكهربائى وارتفاع إنزيمات القلب (كيناز الكرياتين العضلى الدماغى والتروبونين)، لقد تم استبعاد المرضى الذين يعانون من داء السكرى وأمراض الكبد والكلى والغدة الدرقية للقضاء على اى علاقة بين نشاط الانزيم والامراض الاخرى غير أمراض الشريان التاجي.

كما تم اختيار ٢٠ من الافراد الاصحاء ويشملون ٧٠% ذكور، ٣٠% اناث حيث تتراوح اعمارهم من ٣٠- ٦١ عاماً بمتوسط قدره ٤٤ عاماً وليس لهم تاريخ من الداء السكرى ، ارتفاع ضغط الدم والادوية كمجموعة تحكم .

تم جمع ٥ مليلتر من الدم الوريدى من كل فرد عند دخوله المستشفى وتم تفريغه فى انبوب عادى ثم تم تقسيم المصل الى جزئين : الاول تم استخدامه فى قياس المعاملات الكيميائية الاتية (كيناز الكرياتين الكلى – كيناز الكرياتين العضلى الدماغى – التروبونين – نازعه اكاتات و ناقله اسبارتاتى) بواسطة محلل كيمائى (تشخيص الرعاية الصحية سيمنز ، الولايات المتحدة الامريكية ما عدا البروتين المتفاعل ج ذو الحساسية المرتفعة تم قياسه بواسطة محلل كيمائى كوباس سى ٣١١).

اما الجزء الثانى من المصل فقد تم حفظه فى درجة حرارة - ٨٠ درجة مئوية لقياس نشاط الانزيم بواسطة محلل كيمائى بيكمان كولتر.

تم جمع ٣ مليلتر من الدم الوريدى لكل فرد بعد صيام ١٢ ساعة وتم تفريغها فى انبوب عادى تم استخدام المصل لقياس صورة الدهون (الدهون الثلاثية –الكوليستيرول – البروتونات الدهنية منخفضة الكثافة والبروتونات الدهنية عالية الكثافة) والصيام السكر فى الدم بواسطة محلل كيمائى (تشخيص الرعاية الصحية سيمنز، الولايات المتحدة الامريكية).

فى هذه الدراسة وجدنا انه ليس هناك ارتباط احصائي بين المرضى والاصحاء من حيث النوع ، السن والتخين. ومن ناحية اخرى وجدنا ارتباط احصائي بين المرضى والاصحاء من حيث ارتفاع ضغط الدم.

لقد توصلنا الي وجود زيادة في (الدهون الثلاثية –الكوليستيرول –البروتونات الدهنية عالية الكثافة والصيام السكر فى الدم) في المرضى عن الاصحاء مع وجود ارتباط احصائي بين المجموعتين.

فى هذه الدراسة قمنا بقياس العديد من دلالات القلب (كيناز الكرياتين الكلى – كيناز الكرياتين العضلى الدماغى – التروبونين – نازعه اكاتات - ناقله اسبارتاتى و البروتين المتفاعل ج ذو الحساسية المرتفعة) ووجدنا زيادة احصائية فى المرضى عن الاصحاء.

فِي دَرَاستِنَا وَجَدْنَا أَن مَعْدَل نَشَاطِ الْاَنْزِيمِ الْفوسفولِيْبِيَازِ الْمُرْتَبَطِ بِالْبِرُوتِيْنِ الدَّهْنِي ٢أ كَانَ اعْلَى بَيْنَ الْمُرْضِي مَقَارَنَةً مَعَ الْاِصْحَاءِ وَكَانَ مُرْتَبَطًا مَعَ زِيَادَةِ مَعْدَلِ حُدُوثِ احْتِشَاءِ عَضَلَةِ الْقَلْبِ .

لَقَدْ تَحَقَّقْنَا مِنْ عَدَمِ وُجُودِ اِرْتِبَاطِ اِحْصَائِي بَيْنَ نَشَاطِ الْاَنْزِيمِ الْفوسفولِيْبِيَازِ الْمُرْتَبَطِ بِالْبِرُوتِيْنِ الدَّهْنِي ٢أ وَالنَّوْعِ، التَّدْخِيْنِ اَوْ اِرْتِفَاعِ ضَغْطِ الدَّمِ.

فِي هَذِهِ الدَّرَاسَةِ تَحَقَّقْنَا مِنْ اِلْرْتِبَاطِ الْاِحْصَائِي بَيْنَ نَشَاطِ الْاَنْزِيمِ الْفوسفولِيْبِيَازِ الْمُرْتَبَطِ بِالْبِرُوتِيْنِ الدَّهْنِي ٢أ وَدَلَالَاتٍ مُتَعَدَّةٍ. وَوَجَدْنَا اِرْتِبَاطَ اِحْصَائِي اِجْبَائِي بَيْنَ نَشَاطِ الْاَنْزِيمِ الْفوسفولِيْبِيَازِ الْمُرْتَبَطِ بِالْبِرُوتِيْنِ الدَّهْنِي ٢أ وَ (الدَّهُونِ الثَّلَاثِيَّةِ وَ الْبِرُوتُونَاتِ الدَّهْنِيَّةِ مُنْخَفِضَةِ الْكثَافَةِ). لَقَدْ تَوَصَّلْنَا اِلَى عَدَمِ وُجُودِ اِرْتِبَاطِ اِحْصَائِي بَيْنَ (السِّنِّ، الْكُولِيْسْتِيرُولِ، كِيْنَازِ الْكْرِيَاتِيْنِ الْكَلِي - كِيْنَازِ الْكْرِيَاتِيْنِ الْعَضَلِيِّ الدَّمَاغِي - الثَّرُوبُونِيْنِ - نَازَعِ اِكْتَاتِ - نَاقِلِهِ اِسْبَارْتَاتِي وَ الْبِرُوتِيْنِ الْمُتَفَاعِلِ ج ذُو الْحَسَاسِيَّةِ الْمُرْتَفَعَةِ) وَنَشَاطِ الْاَنْزِيمِ الْفوسفولِيْبِيَازِ الْمُرْتَبَطِ بِالْبِرُوتِيْنِ الدَّهْنِي ٢أ بَيْنَ الْمُرْضِي. كَمَا كَانَ هُنَاكَ اِرْتِبَاطَ اِحْصَائِي عَكْسِي بَيْنَ نَشَاطِ الْاَنْزِيمِ الْفوسفولِيْبِيَازِ الْمُرْتَبَطِ بِالْبِرُوتِيْنِ الدَّهْنِي ٢أ وَ الْبِرُوتُونَاتِ الدَّهْنِيَّةِ عَالِيَةِ الْكثَافَةِ بَيْنَ الْمُرْضِي.

اٰخِيْرًا لَمْ نَلَاظْ اِي اِرْتِبَاطَ اِحْصَائِي بَيْنَ نَشَاطِ الْاَنْزِيمِ الْفوسفولِيْبِيَازِ الْمُرْتَبَطِ بِالْبِرُوتِيْنِ الدَّهْنِي ٢أ وَايٍ مِنْ الدَّلَالَاتِ السَّابِقَةِ بَيْنَ الْاِصْحَاءِ.

قياس نشاط انزيم الفسفوليبياز ٢ المرتبط بالبروتين الدهنى فى مصل مرضى احتشاء عضلة القلب

رسالة علمية

مقدمة لكلية الطب – جامعة الإسكندرية
إيفاءً جزئياً لشروط الحصول على درجة

الماجستير فى الباثولوجيا الإكلينيكية والكيميائية

مقدمة من

ياسمين نبيه إسماعيل زهران

بكالوريوس الطب والجراحة – جامعة الإسكندرية

كلية الطب
جامعة الإسكندرية
٢٠١٥

قياس نشاط انزيم الفسفوليبياز ٢ المرتبط بالبروتين الدهنى فى مصل مرضى احتشاء عضلة القلب

مقدمة من

ياسمين نبيه اسماعيل زهران

بكالوريوس الطب والجراحة- الإسكندرية

للحصول على درجة

الماجستير فى الباثولوجيا الإكلينيكية والكيميائية

موافقون

لجنة المناقشة والحكم على الرسالة

.....

أ.د/ وفاء سعد رجب

أستاذ الباثولوجيا الكيميائية

معهد البحوث الطبية

جامعة الإسكندرية

.....

أ.د/ علا عاطف شراكى

أستاذ الباثولوجيا الإكلينيكية والكيميائية

كلية الطب

جامعة الإسكندرية

.....

أ.د/ بسنت السيد معز

أستاذ الباثولوجيا الإكلينيكية والكيميائية

كلية الطب

جامعة الإسكندرية

التاريخ:

السادة المشرفون

.....

أ.د/ علا عاطف شراكي

أستاذ الباثولوجيا الإكلينيكية والكيميائية

كلية الطب

جامعة الإسكندرية

.....

أ.د/ محمد أحمد صبحي

أستاذ أمراض القلب والأوعية الدموية

كلية الطب

جامعة الإسكندرية

.....

أ.د/ عبلة أحمد أبو زيد

أستاذ الباثولوجيا الإكلينيكية والكيميائية

كلية الطب

جامعة الإسكندرية