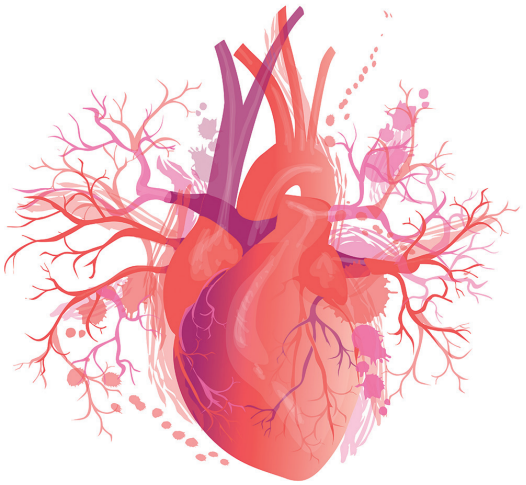

BÖLÜM 11

MİYOKARDİYAL KORUNMA

Veysel YAVUZ[1]

GİRİŞ

Miyokardiyal koruma, reperfüzyon hasarı ile birlikte yüksek riskli cerrahi sonrası kardiyak morbidite ve mortaliteden başlıca sorumlu olan, kalbin iskemik hasara dayanma yeteneğini artıran, yöntemleri ifade eder. Açık kalp cerrahisinin başladığı ilk yıllardan beri, morbidite ve mortalitenin büyük oranda postoperatif kardiyak pompa yetersizliği ile ilgili olduğu dikkati çekmiştir.

TARİHÇE

Miyokard koruması ile ilgili ilk metodun kim tarafından düşünüldüğü kesin bilinmemekle birlikte 1956'da Lillehei ve arkadaşları tarafından yapılan aort kapak cerrahisinde, retrograd koroner perfüzyon tekniği ilk metot olarak kabul edilebilir (1). Swan ve arkadaşları tarafından 1953'te hipotermik arrest ve Melrose ve arkadaşları tarafından 1955 yılında ortaya sürülen elektif kimyasal kardiyak arrest önerileri miyokardı korumaktan ziyade, daha rahat ve kansız operasyon alanı amaçladıkları için ilk metotlar olarak kabul edilmemesi daha doğru bir yaklaşımdır (2,3). Daha sonra farklı yöntemler ve teknikler araştırılmaya devam edilerek miyokard hasarın daha iyi anlaşılmasına ışık tutmuş ve bu sayede iskemik hasar, nekroz, stone heart ve stunning kelimeleri daha anlaşılır hale gelmiştir.

MİYOKARD REPERFÜZYONUNUN PATOFİZYOLOJİSİ

İskemi, vücuttaki herhangi bir doku ya da organa kan akışının azalması veya kesilmesine bağlı olarak gelişen enerji sunumu ve talebi arasındaki dengesizliği ifade etmektedir. Yeterli süreli iskemi hücre ölümüyle sonuçlanır. Bununla birlikte, iskemik saldırı uygun bir noktada kesintiye uğrarsa bile, hasta aritmiler ve düşük kalp debisi durumu dâhil olmak üzere çeşitli zararlı sekeller ile boğuşan canlı bir miyokard ile baş başa kalacaktır. Bu doğrudan reperfüzyon hasarının sonucudur. Kesin hücre ölümü önlenirse, bundan sonra hücre için iki olası alternatif yol vardır, bunlar stunning ve hiberanasyondur.

Koroner arter operasyonu geçiren hastalarda miyokard dokusu, koronerlere gelen kan akımı veya miyokardın ya da her ikisinin birden anormal olduğu durumlarda iskemi reperfüzyon hasarına karşı daha duyarlı hale gelir. Hipertrofik

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sinimi olan hastaların ve iskemik kalp hastalığı riski taşıyan hastaların kalp hızının 50-60 atım/dk olacak şekilde titre edilmesini önermektedir.

KAYNAKLAR

1. Lillehei CW, Dewall Ra, Gott Vl, Varco Rl.The direct vision correction of calcific aortic stenosis by means of a pump-oxygenator and retrograde coronary sinus perfusion.Dis Chest 1956; 30:123-132
2. Swan H, Zeavın I, Blount Sg Jr, Virtue RW. Surgery by direct vision in the open heart during hypothermia.J Am Med Assoc 1953: 21;153:1081-1085
3. Melrose DG, Dreyer B, Bentall HH, Baker JB. Elective cardiac arrest.Lancet 1955:2;269:21-22
4. Schaper J, Scheld HH, Schmidt U, Hehrlein F. Ultrastructural study comparing the efficacy of five different methods of intraoperative myocardial protection in the human heart. J Thorac Cardiovasc Surg 1986; 92:47-55
5. Batist G, Mersereau W, Malashenko BA, Chiu RC. Response to ischemia-reperfusion injury in hypertrophic heart, role of free-radical metabolic pathways. Circulation 1989; 80:10-13
6. Julia P, Kofsky ER, Buckberg GD, Young HH, Bugyi HI. Studies of myocardial protection in the immature heart. models of ischemic and hypoxic/ischemic injury in the immature puppy heart.J Thorac Cardiovasc Surg 1991;101:14-22
7. Ellis SG, Henschke CI, Sandor T, Wynne J, Braunwald E, Kloner RA. Time course of functional and biochemical recovery of myocardium salvaged by reperfusion.J Am Coll Cardiol 1983;1: 1047-1055
8. Braunwald E, Kloner RA. The stunned myocardium: prolonged, postischemic ventricular dysfunction. Circulation 1982;66: 1146-1149
9. Bolli R, Marbán E. Molecular and cellular mechanisms of myocardial stunning. Physiol Rev 1999; 79:609-634
10. Piper HM, García-Dorado D. Prime causes of rapid cardiomyocyte death during reperfusion. Ann Thorac Surg 1999; 68:1913-1919
11. Verbunt RJ, Van Der Laarse A. Glutathione metabolism in nonischemic and postischemic rat hearts in response to an exogenous prooxidant. Mol Cell Biochem 1997;167:127-134
12. Sharikabad MN, Hagelin EM, Hagberg IA, Lyberg T, Brørs O. Effect of calcium on reactive oxygen species in isolated rat cardiomyocytes during hypoxia and reoxygenation. J Mol Cell Cardiol 2000; 32:441-452
13. Bolli R, Patel BS, Hartley CJ, Thornby JI, Jeroudi MO, Roberts R. Nonuniform transmural recovery of contractile function in stunned myocardium. Am J Physiol 1989;257:H375-385
14. Li Q, Bolli R, Qiu Y, Tang XL, Murphree SS, French BA. Gene therapy with extracellular superoxide dismutase attenuates myocardial stunning in conscious rabbits. Circulation 1998; 98:1438-1448
15. Miyata H, Lakatta EG, Stern MD, Silverman HS. Relation of mitochondrial and cytosolic free calcium to cardiac myocyte recovery after exposure to anoxia. Circ Res 1992; 71:605-613
16. Tanaka M, Richard VJ, Murry CE, Jennings RB, Reimer KA. Superoxide dismutase plus catalase therapy delays neither cell death nor the loss of the ttc reaction in experimental myocardial infarction in dogs. J Mol Cell Cardiol 1993; 25:367-378
17. Rahimtoola SH, Griffith GC. The hibernatig myocardium Am Heart J 1989; 117-211
18. Braunwald E. Myocardial reperfusion, limitation of infarct size, reduction of left ventricular dysfunction, and improved survival. should the paradigm be expanded? Circulation 1989; 79:441-444
19. Cobbe Sm, Poole-Wilson Pa. The time of onset and severity of acidosis in myocardial ischaemia.J Mol Cell Cardiol 1980;12:745-760
20. Garlick Pb, Radda Gk, Seeley Pj. Studies of acidosis in the ischaemic heart by phosphorus nuclear magnetic resonance. Biochem J 1979; 15;184:547-554
21. Kerr JFR, Wyllie AH, Currie AR. Apoptosis: A basic biological phenomenon with wide-ranging implications in tissue kinetics. Br J Cancer 1972 26:239-257
22. Watanabe BI, Premaratne S, Limm W, Mugiishi MM, McNamara JJ. High- and low-dose superoxide dismutase plus catalase does not reduce myocardial infarct size in a subhuman primate model. Am Heart J 1993;126:840-846
23. Maulik N, Yoshida T, Das DK. Oxidative stress developed during the reperfusion of ischemic myocardium induces apoptosis. Free Radic Biol Med 1998; 24:869-875
24. Van Heerde Wl, Robert-Offerman S, Dumont E, Hofstra L, Doevendans PA, Smits JF, Daemen MJ, Reutelingsperger CP. Markers of apoptosis in cardiovascular tissues: focus on Annexin V. Cardiovasc Res 2000; 45:549-559

25. Hammill AK, Uhr JW, Scheuermann RH. Annexin V staining due to loss of membrane asymmetry can be reversible and precede commitment to apoptotic death. Exp Cell Res 1999; 251: 16-21
26. Buckberg G. Myocardial temperature management during aortic clamping for cardiac surgery. J Thorac Cardiovasc Surg 1991; 102: 895–903
27. Newman M. Perioperative Organ Protection. Baltimore: Lippincott Williams & Wilkins, 2003
28. Veena G, Omura J, Alghamdi A, Weisel R, Fremes S. Is blood superior to crystalloid cardioplegia A meta-analysis of randomized clinical trials. Circulation 2006; 114: 331–8
29. Murray CE, Jennings RB, Reimer KA. Preconditioning with ischemia: a delay of lethal cell injury in ischemic myocardium. Circulation 1986; 74:1124-1136
30. Zaugg M, Lucchinetti E. Cardiac (Pre)Conditioning: Concepts, Mechanisms, Perspectives. IARS 2007 Review Course Lectures
31. Cohen MV, Baines CP, Downey JM. Ischemic preconditioning: From adenosine receptor of KATP channel. Annu Rev Physiol 2000; 62:79-109
32. Yellon DM, Baxter GF, Garcia-Dorado D, Heusch G, Sumeray MS. Ischaemic preconditioning:Present position and future directions. Cardiovasc Res 1998; 37:21-33
33. Scognamiglio R, Avogaro A, Vigili dK et al. Effects of treatment with sulfonylurea drugs or insulin on ischemia-induced myocardial dysfunction in type 2 diabetes. Diabetes 2002; 51: 808–12
34. Bolli R.The late phase of preconditioning. Circ Res 2000; 87:972-83
35. Stein AB, Bolli R, Guo Yet al. The late phase of ishaemic preconditioning induces a prosurvival genetic program that results in marked attenuation of apoptosis. J Mol Cell Cardiol 2007; 42: 1075–85
36. Okishige K, Yamashita K, Yoshinaga HAzegami K, Satoh T, Goseki Y, Fujii S, Ohira H, Satake S. Electrophysiologic effects of ischemic preconditioning on QT dispersion during coronary angioplasty. J Am Coll Cardiol 1996; 28:70-73
37. Jneid H, Leessar M, Bolli R. Cardiac preconditioning during percutaneous coronary interventions. Cardiovasc Drugs Ther 2005; 19:211-217
38. Lasley RD, Keith BJ, Kristo G, Yoshimura Y, Mentzer RM Jr. Delayed adenosine A1 receptor preconditioning in rat myocardium is MAPK dependent but iNOS independent. Am J Physiol Heart Circ Physiol 2005; 289:H785-791
39. Ali Z, Callaghan C, Lim E et al. Remote ischaemic preconditioning reduces myocardial and renal injury after elective abdominal aorticaneurysm repair: a randomized controlled trial. Circulation 2007; 11: I-98–105
40. Ten Broecke P, De Hert S, Mertens E, Adriaensen. Effect of preoperative b-blockade on perioperative mortality in coronary surgery. Br J Anaesth 2003; 90: 27–311
41. Biccard B, Sear J, Foe¨x P. Statin therapy: a potentially useful perioperative intervention in patients with cardiovascular disease. Anaesthesia 2005; 60: 1106–14
42. Liu S, Block B, Wu C. Effects of perioperative central neuraxial analgesia on outcome after coronary artery bypass surgery. Anesthesiology 2004; 101: 153–61