Neopstruktivna koronarna bolest – etiopatogeneza i predložena klinička klasifikacija poremećaja

Nonobstructive coronary artery disease – etiopathogenesis and suggested clinical classification of the disorder

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Godine 1967. učinjena je prva perkutana selektivna angiografija. Iste godine, točno 10 godina prije prve perkutane koronarne intervencije (PCI), opisan je prvi bolesnik s anginom pectoris i urednom angiografijom koronarnih arterija. Od tada, gotovo punih 50 godina, klinički entitet neopstruktivne koronarne bolesti srca (KBS) ne prestaje biti predmet rasprave i istraživanja.¹⁻³ Dvije su osnovne grupe hipoteza koje nastoje objasniti patofiziološke mehanizme neopstruktivne KBS: ishemijska i neishemijska grupa hipoteza. Ishemijska teorija se osniva na abnormalnoj mikrovaskularnoj funkciji, a neishemijska teorija na promijenjenoj percepciji boli. Ishemijska hipoteza obuhvaća čimbenike koji mogu biti endotel ovisni ili neovisni. Neishemijska hipoteza promijenjene percepcije boli dijeli se prema zahvaćenosti neuralnog sustava na aferentnu ili eferentnu komponentu. U pozadini ishemijske, endotel ovisne disfunkcije nalaze se arterijska hipertenzija, šećerna bolest, intolerancija glukoze, dislipidemija te pušenje, a u pozadini endotel neovisne disfunkcije nalazi se upala, manjak estrogena u žena, histerektomija i inzulinska rezistencija. Neishemijsku grupu hipoteza sačinjavaju teorije neuralnog poremećaja, koji mogu biti aferentna ili eferentna- nociceptivna te teorija habitualnog poremećaja (bihevioristički pristup). Postoji veza između čimbenika prikazanih u sklopu mikrovaskularne disfunkcije i neishemijskih čimbenika. Smatra se da ponavljane epizode mikroishemije mogu putem fibroze i blaže prolongirane inflamacije dovesti do razvoja neuralne abnormalnosti na aferentnoj i eferentnoj razini. Tako je zatvoren krug koji patofiziološki povezuje mikrovaskularnu disfunkciju i neuralni poremećaj u neopstruktivnoj KBS.

In 1967 a first case of angina with angiographically normal coronary vessels was described. That same year, exactly 10 years before the first percutaneous coronary intervention (PCI), the first patient with angina and normal coronary angiography was described. Ever since, for nearly 50 years, this clinical entity remains a subject of numerous discussions and studies.¹⁻³ There are two basic groups of hypothesis to explain the pathophysiological mechanisms of non-obstructive coronary artery disease (CAD): ischemic and non-ischemic groups of hypothesis. Ischemic theory is based on abnormal microvascular function, and non-ischemic theory is based on the changed perception of pain. Ischemic hypothesis includes the factors that may be endothelium-dependent or independent. Non-ischemic hypothesis is divided according to the involvement of the neural system in the afferent and efferent components. In the background of ischemic, endothelium dependent dysfunction are hypertension, diabetes, glucose intolerance, dyslipidemia, smoking, and in the background of endothelium dysfunction independent hypothesis is inflammation, lack of estrogen in women, hysterectomy, insulin resistance. Non-ischemic group consists hypothesis theory of neural disorders which may be afferent-adrenergic or efferent-nociceptive, and theory of habitual disorders (behavioral approach). There is a connection between factors which are displayed in microvascular dysfunction and nonischemic factors. It is believed that repeated episodes of microischemia by fibrosis and less severe prolonged inflammation lead to the development of neural abnormalities on afferent and efferent level. Thus, there are closed circuit that connects pathophysiological microvascular dysfunction and neural disorder in non-obstructive CAD.

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