Protección miocárdica

myocardial protection: Latest results from PubMed
URL: https://pubmed.ncbi.nlm.nih.gov/rss-feed/?feed_id=1lKnPBUS1ZVgVcAw3tPvoYngl6sK7K6o7ePuJpr6j_daulok04&utm_source=Other&utm_medium=rss&ff=20221229103800&v=2.17.9.post6+86293ac&utm_content=1lKnPBUS1ZVgVcAw3tPvoYngl6sK7K6o7ePuJpr6j_daulok04
Actualizado: hace 1 año 12 semanas

Ablation of CXCR4 expression in cardiomyocytes exacerbates isoproterenol-induced cell death and heart failure

Jue, 12/29/2022 - 11:00

Int J Mol Med. 2023 Feb;51(2):13. doi: 10.3892/ijmm.2022.5216. Epub 2022 Dec 29.

ABSTRACT

CXCR4 is a seven-transmembrane-spanning Gi-coupled receptor for the SDF-1 chemokine and plays a critical role in cardiovascular development and post-injury repair. However, the specific role of CXCR4 in cardiomyocytes is incompletely understood. It was hypothesized that CXCR4 activation in cardiomyocytes antagonizes β-adrenoceptor/Gs signaling-induced cardiac dysfunction. Cardiomyocyte-specific CXCR4 knockout (CXCR4-CMKO) mice were generated by crossing CXCR4fl/fl and MHC-Cre+/- mice. Their cardiac structure and function in the basal state are equivalent to that of the control MHC-Cre+/- littermates until at least 4 months old. However, following continuous subcutaneous administration of isoproterenol (Iso) via an osmotic mini-pump, the ventricular myocardial contractility, dilation, cardiomyocyte apoptosis, and interstitial fibrosis are worse in CXCR4-CMKO mice than in MHC-Cre+/- littermates. In the cultured H9C2 cardiomyocytes, SDF-1 treatment markedly attenuated Iso-induced apoptosis and reduction in phospho-Akt, and this protective effect was lost by knockdown of CXCR4 or by co-treatment with Gi inhibitors. In conclusion, CXCR4 promotes cardiomyocyte survival and heart function during β-adrenergic stress.

PMID:36579657 | DOI:10.3892/ijmm.2022.5216

Categorías: Protección miocárdica

Emergency Care in the Occupied Palestinian Territory: A Scoping Review

Jue, 12/29/2022 - 11:00

Health Hum Rights. 2022 Dec;24(2):255-263.

ABSTRACT

The development of robust emergency care systems as a critical platform for addressing the global burden of disease has been increasingly recognized by global health policy makers over the past decade. A human rights-based approach to securing the right to quality emergency care is also essential to respond to the structural and political determinants of poor health outcomes. In the occupied Palestinian territory, human rights violations have contributed to significant deficiencies in health and quality of health care. In this scoping review, we identify deficiencies in the management of high-risk presentations to emergency departments in the Palestinian health care system for traumatic injury, acute myocardial infarction, and stroke. We subsequently apply a human rights-based analysis to demonstrate how structural racism in the administration of the occupation has contributed to deficiencies in emergency care. Specifically, deficiencies in resource and system organization within the Palestinian emergency care system arise due to occupation-related

restrictions on freedom of movement, the procurement of essential drugs and medical equipment, and the development of a national Palestinian health care system. Further research and intervention are needed to understand gaps in emergency care for Palestinians and, in turn, to improve the management of emergency medical and traumatic conditions through capacity building of a Palestinian emergency care system. Importantly, deconstruction of the structural determinants of poor health for Palestinians in the occupied territory is needed to improve public health and ensure the protection of human rights.

PMID:36579300 | PMC:PMC9790939

Categorías: Protección miocárdica

Single coronary ostium with obstructive hypertrophic cardiomyopathy treated using the Morrow procedure: a case report

Mié, 12/28/2022 - 11:00

J Cardiothorac Surg. 2022 Dec 28;17(1):340. doi: 10.1186/s13019-022-02084-2.

ABSTRACT

BACKGROUND: Hypertrophic cardiomyopathy is a commonly inherited heart disease. In addition, single coronary artery (SCA) is a rare congenital anomaly of the coronary arteries. And SCA concomitant with severe hypertrophic obstructive cardiomyopathy (HOCM) has seldom been reported in the literature. However, such cases have not been reported to be treated with the Morrow procedure.

CASE PRESENTATION: Herein, we presented a case of a 64-year-old female diagnosed with a single left coronary artery with severe HOCM. The HOCM was treated with the Morrow procedure. The patient was discharged on the seventh postoperative day and was asymptomatic during the follow-up.

CONCLUSION: To our knowledge, this is the first study reporting a single left coronary artery with severe HOCM treated with the Morrow procedure. In addition, myocardial protection by cardioplegia antegrade perfusion was safe for the patient with SCA and HOCM.

PMID:36578088 | DOI:10.1186/s13019-022-02084-2

Categorías: Protección miocárdica

Resveratrol Attenuates Sepsis-Induced Cardiomyopathy in Rats through Anti-Ferroptosis via the Sirt1/Nrf2 Pathway

Mié, 12/28/2022 - 11:00

J Invest Surg. 2023 Dec;36(1):2157521. doi: 10.1080/08941939.2022.2157521.

ABSTRACT

Background: Sepsis-induced cardiomyopathy (SIC) is a severe myocardial dysfunction secondary to septicemia. It is a major concern owing to the high mortality and morbidity, which are greatly influenced by ferroptosis. Resveratrol (RSV) is a naturally existing agonist of the silent information regulator 1 (Sirt1). It has cardioprotective effects against sepsis-induced myocardial injury, However, the detailed mechanism is unknown.**Methods:** In this study, cecal ligation and puncture (CLP)-induced septic rats were employed to assess the changes in ferroptosis with RSV administration. According to the different treatments the rats were divided into the following groups: (1) the Sham, (2) CLP, (3) CLP + RSV at various doses (10, 30, and 50 mg/kg), and (4) CLP + Fer-1(a

ferroptotic inhibitor) groups. After 24 h, the structure and function of the cardiac system in rats were evaluated, and mitochondrial morphology, ferroptosis-related biomarkers, and the levels of Sirt1/Nrf2 were assessed.**Results:** The rats that underwent CLP had suffered cardiac dysfunction, accompanied with myocardial damage, impaired mitochondria, elevated lipid peroxidation, and reduced Sirt1/Nrf2 expression in the myocardium. High-dose RSV successfully improved heart function, reversing the abnormalities in a dose-dependent manner. We then used EX527, a selective Sirt1 inhibitor, to further identify the intermediate signaling targets of RSV that regulate ferroptosis. EX527 diminished the curative effects of high-doses RSV.**Conclusions:** Summarily, our findings suggest a novel mechanism of RSV in reducing SIC: ferroptosis inhibition via upregulation of Sirt1/Nrf2 signaling pathways. This may be an effective therapeutic approach against organ failure in sepsis, particularly SIC.

PMID:36576230 | DOI:10.1080/08941939.2022.2157521

Categorías: Protección miocárdica

Hydrogen Sulfide Modulates Endothelial-Mesenchymal Transition in Heart Failure

Mié, 12/28/2022 - 11:00

Circ Res. 2022 Dec 28. doi: 10.1161/CIRCRESAHA.122.321326. Online ahead of print.

ABSTRACT

BACKGROUND: Hydrogen sulfide is a critical endogenous signaling molecule that exerts protective effects in the setting of heart failure. Cystathionine γ-lyase (CSE), 1 of 3 hydrogen-sulfide-producing enzyme, is predominantly localized in the vascular endothelium. The interaction between the endothelial CSE-hydrogen sulfide axis and endothelial-mesenchymal transition, an important pathological process contributing to the formation of fibrosis, has yet to be investigated.

METHODS: Endothelial-cell-specific CSE knockout and Endothelial cell-CSE overexpressing mice were subjected to transverse aortic constriction to induce heart failure with reduced ejection fraction. Cardiac function, vascular reactivity, and treadmill exercise capacity after transverse aortic constriction were measured to determine the severity of heart failure. Histological and gene expression analyses were performed to investigate changes in cardiac fibrosis and endothelial-mesenchymal transition activation.

RESULTS: Endothelial-cell-specific CSE knockout mice exhibited increased endothelial-mesenchymal transition and reduced nitric oxide bioavailability in the myocardium, which was associated with increased cardiac fibrosis, impaired cardiac and vascular function, and worsened exercise performance. In contrast, genetic overexpression of CSE in endothelial cells led to increased myocardial nitric oxide, decreased endothelial-mesenchymal transition and cardiac fibrosis, preserved cardiac and endothelial function, and improved exercise capacity.

CONCLUSIONS: Our data demonstrate that endothelial CSE modulates endothelial-mesenchymal transition and ameliorate the severity of pressure-overload-induced heart failure, in part, through nitric oxide-related mechanisms. These data further suggest that endothelium-derived hydrogen sulfide is a potential therapeutic for the treatment of heart failure with reduced ejection fraction.

PMID:36575984 | DOI:10.1161/CIRCRESAHA.122.321326

Categorías: Protección miocárdica

A RGS7-CaMKII complex drives myocyte-intrinsic and myocyte-extrinsic mechanisms of chemotherapy-induced cardiotoxicity

Mar, 12/27/2022 - 11:00

Proc Natl Acad Sci U S A. 2023 Jan 3;120(1):e2213537120. doi: 10.1073/pnas.2213537120. Epub 2022 Dec 27.

ABSTRACT

Dose-limiting cardiotoxicity remains a major limitation in the clinical use of cancer chemotherapeutics. Here, we describe a role for Regulator of G protein Signaling 7 (RGS7) in chemotherapy-dependent heart damage, the demonstration for a functional role of RGS7 outside of the nervous system and retina. Though expressed at low levels basally, we observed robust up-regulation of RGS7 in the human and murine myocardium following chemotherapy exposure. In ventricular cardiomyocytes (VCM), RGS7 forms a complex with Ca2+/calmodulin-dependent protein kinase (CaMKII) supported by key residues (K412 and P391) in the RGS domain of RGS7. In VCM treated with chemotherapeutic drugs, RGS7 facilitates CaMKII oxidation and phosphorylation and CaMKII-dependent oxidative stress, mitochondrial dysfunction, and apoptosis. Cardiac-specific RGS7 knockdown protected the heart against chemotherapy-dependent oxidative stress, fibrosis, and myocyte loss and improved left ventricular function in mice treated with doxorubicin. Conversely, RGS7 overexpression induced fibrosis, reactive oxygen species generation, and cell death in the murine myocardium that were mitigated following CaMKII inhibition. RGS7 also drives production and release of the cardiokine neuregulin-1, which facilitates paracrine communication between VCM and neighboring vascular endothelial cells (EC), a maladaptive mechanism contributing to VCM dysfunction in the failing heart. Importantly, while RGS7 was both necessary and sufficient to facilitate chemotherapy-dependent cytotoxicity in VCM, RGS7 is dispensable for the cancer-killing actions of these same drugs. These selective myocyte-intrinsic and myocyte-extrinsic actions of RGS7 in heart identify RGS7 as an attractive therapeutic target in the mitigation of chemotherapy-driven cardiotoxicity.

PMID:36574707 | DOI:10.1073/pnas.2213537120

Categorías: Protección miocárdica

Glimepiride Use is Associated with Reduced Cardiovascular Mortality in Patients with Type 2 Diabetes and Chronic Heart Failure: A Prospective Cohort Study

Mar, 12/27/2022 - 11:00

Eur J Prev Cardiol. 2022 Dec 27:zwac312. doi: 10.1093/eurjpc/zwac312. Online ahead of print.

ABSTRACT

BACKGROUND: Glimepiride has good cardiovascular safety. However, whether glimepiride benefits clinical cardiovascular outcomes is unclear.

METHODS: A total of 21,451 inpatients with type 2 diabetes (T2D) and chronic heart failure (CHF) were analyzed, including 638 who received glimepiride treatment and 20,813 who did not. Propensity score matching yielded 509 pairs (glimepiride and non-glimepiride groups), and both groups were followed up. Kaplan-Meier and Cox regression analyses were used to compare all-cause mortality, cardiovascular mortality, hospitalizations and emergency visits for heart failure, and hospitalizations for acute myocardial infarction or stroke.

RESULTS: During follow-up, the all-cause mortality (adjusted hazard ratio [HR], 0.47; 95% confidence interval [CI], 0.35-0.63; P < 0.001), cardiovascular mortality (adjusted HR, 0.34; 95% CI, 0.24-0.48; P < 0.001), and number of hospitalizations and emergency visits for heart failure (adjusted HR, 0.42; 95% CI, 0.36-0.50; P < 0.001) and hospitalizations for acute myocardial infarction or stroke (adjusted HR, 0.53; 95% CI, 0.38-0.73; P < 0.001) were significantly lower in the glimepiride group; the conclusion remained similar in all subgroups. Furthermore, high-dose glimepiride use (2-4 mg/day) was associated with lower cardiovascular mortality than low-dose (1 mg/day) (adjusted HR, 0.55; 95% CI, 0.31-0.99; P = 0.047). Glimepiride exhibited good molecular docking with soluble epoxide

hydrolase (sEH) and increased the level epoxyeicosatrienoic acid (EET).

CONCLUSIONS: Long-term continuous glimepiride use is associated with better survival, fewer hospitalizations and emergency visits for heart failure, and fewer hospitalizations for acute myocardial infarction or stroke in patients with T2D and CHF. High-dose glimepiride has greater cardiovascular protective advantages than low-dose glimepiride. The cardiovascular protective effect of glimepiride may be related to the EET level increase through sEH inhibition.

PMID:36573717 | DOI:10.1093/eurjpc/zwac312

Categorías: Protección miocárdica

Biomaterials-based Cell Therapy for Myocardial Tissue Regeneration

Lun, 12/26/2022 - 11:00

Adv Healthc Mater. 2022 Dec 26:e2202699. doi: 10.1002/adhm.202202699. Online ahead of print.

ABSTRACT

Cardiovascular diseases have been the leading cause of death worldwide during the past several decades. Cell loss is the main problem that resulted in cardiac dysfunction and further mortality. Cell therapy aiming to replenish the lost cells is proposed to treat cardiovascular diseases especially ischemic heart diseases which lead to a big portion of cell loss. Due to the direct injection's low cell retention and survival ratio, cell therapy using biomaterials as cell carriers attracts more and more attention because of their promotion of cell delivery and maintenance at the aiming sites. In this review, we systematically summarized the three main factors involved in cell therapy for myocardial tissue regeneration: cell sources (somatic cells, stem cells and engineered cells), chemical components of cell carriers (natural materials, synthetic materials and electroactive materials), and categories of cell delivery materials (patches, microspheres, injectable hydrogels, nanofiber and microneedles, etc.). An introduction of the methods including magnetic resonance/radionuclide/photoacoustic and fluorescence imaging for tracking the behavior of transplanted cells in vivo is also included. Current challenges of biomaterials-based cell therapy and their future directions are provided to give both beginners and professionals a clear view of the development and future trends in this area.

PMID:36572412 | DOI:10.1002/adhm.202202699

Categorías: Protección miocárdica

Ginsenoside Rh2 attenuates myocardial ischaemia-reperfusion injury by regulating the Nrf2/HO-1/NLRP3 signalling pathway

Lun, 12/26/2022 - 11:00

Exp Ther Med. 2022 Nov 29;25(1):35. doi: 10.3892/etm.2022.11734. eCollection 2023 Jan.

ABSTRACT

Ginsenoside Rh2 (GRh2) is a monomer isolated from red ginseng that has extensive pharmacological effects. However, whether GRh2 has a protective effect on ischaemia/reperfusion (I/R) in the myocardium has yet to be elucidated. The present study aimed to identify the anti-inflammatory and antioxidant effects of GRh2 on I/R in the myocardium and its underlying mechanism. A rat model of myocardial I/R injury was constructed by ligating the left anterior descending coronary artery, which was subsequently treated with GRh2. A total of 40 male Sprague-Dawley rats were divided into the following four groups: The sham group, the I/R group, the I/R+GRh2 (10 mg/kg) group and the I/R+GRh2 (20 mg/kg) group. Neonatal rat cardiomyocytes were also used to evaluate the protective

effect of GRh2 on hypoxia/reoxygenation (H/R)-induced myocardial injury *in vitro*. The GRh2 pre-treatment reduced the I/R- or H/R-induced release of myocardial enzymes and the production of IL-1β, IL-18 and TNF-α. GRh2 reduced the area of myocardial infarction and the histological changes in the myocardium and improved cardiac functions. In addition, GRh2 reduced the expression levels of NOD-like receptor family pyrin domain-containing 3 (NLRP3), apoptosis-associated speck-like protein, caspase-1, malondialdehyde and reactive oxygen species and increased the expression levels of nuclear factor E2-related factor 2 (Nrf2), heme oxygenase-1 (HO-1), glutathione peroxidase and superoxide dismutase. In conclusion, the present study confirmed that GRh2 could reduce oxidative stress and inflammation in cardiomyocytes after reperfusion, and its mechanism of action may be related to its regulation of the Nrf2/HO-1/NLRP3 signalling pathway.

PMID:36569435 | PMC:PMC9764046 | DOI:10.3892/etm.2022.11734

Categorías: Protección miocárdica

Activation of Nrf2 signaling: A key molecular mechanism of protection against cardiovascular diseases by natural products

Lun, 12/26/2022 - 11:00

Front Pharmacol. 2022 Dec 8;13:1057918. doi: 10.3389/fphar.2022.1057918. eCollection 2022.

ABSTRACT

Cardiovascular diseases (CVD) are a group of cardiac and vascular disorders including myocardial ischemia, congenital heart disease, heart failure, hypertension, atherosclerosis, peripheral artery disease, rheumatic heart disease, and cardiomyopathies. Despite considerable progress in prophylaxis and treatment options, CVDs remain a leading cause of morbidity and mortality and impose an extremely high socioeconomic burden. Oxidative stress (OS) caused by disequilibrium in the generation of reactive oxygen species plays a crucial role in the pathophysiology of CVDs. Nuclear erythroid 2-related factor 2 (Nrf2), a transcription factor of endogenous antioxidant defense systems against OS, is considered an ideal therapeutic target for management of CVDs. Increasingly, natural products have emerged as a potential source of Nrf2 activators with cardioprotective properties and may therefore provide a novel therapeutic tool for CVD. Here, we present an updated comprehensive summary of naturally occurring products with cardioprotective properties that exert their effects by suppression of OS through activation of Nrf2 signaling, with the aim of providing useful insights for the development of therapeutic strategies exploiting natural products.

PMID:36569290 | PMC:PMC9772885 | DOI:10.3389/fphar.2022.1057918

Categorías: Protección miocárdica

Effects of different low-dose of insulin glargine on antioxidation of organs in burned rats with delayed resuscitation

Lun, 12/26/2022 - 11:00

Zhonghua Wei Zhong Bing Ji Jiu Yi Xue. 2022 Nov;34(11):1138-1143. doi: 10.3760/cma.j.cn121430-20220627-00604.

ABSTRACT

OBJECTIVE: To study the antioxidant protective effects of different low-dose of insulin glargine on organs of burned rats with delayed resuscitation.

METHODS: Forty male Sprague-Dawley (SD) rats were randomly divided into sham group, delayed resuscitation control group, and insulin glargine 0.5, 1.0, and 2.0 U groups, with 8 rats in each group.

The rats were immersed in hot water (95.0±0.5) centigrade for 15 s to establish the third-degree scald model with 30% total body surface area. The rats in the sham group were immersed in a 37 centigrade water bath for 15 s. Insulin glargine (0.5, 1.0, 2.0 U×kg-1×d-1) was injected subcutaneously in corresponding insulin glargine group 2 hours after injury, and the same amount of normal saline was injected intraperitoneally in the delayed resuscitation control group. Intraperitoneal injection of normal saline 40 mL/kg simulated delayed resuscitation 6 hours after injury in all groups. Abdominal aortic blood samples, heart and kidney tissue were collected immediately after simulating burn in the sham group, and 24 hours after burn in other four groups. The blood glucose, myocardial enzymes [lactate dehydrogenase (LDH), creatine kinase (CK), α-hydroxybutyrate dehydrogenase (α-HBDH), and aspartate aminotransferase (AST)] and renal function indexes [blood urea nitrogen (BUN) and serum creatinine (SCr)] were measured by spectrophotometry, and the isoenzyme MB of creatine kinase (CK-MB) level was determined by immunosuppression method to evaluate the effects of different low-dose insulin glargine intervention on blood glucose, cardiac and renal functions in scalded rats with delayed resuscitation. The oxidative and antioxidant indices [xanthine oxidase (XOD), myeloperoxidase (MPO), copper-zinc superoxide dismutase (CuZn-SOD), catalase (CAT), glutathione peroxidase (GSH-Px), and total antioxidant capacity (T-AOC)] from the heart and kidney tissues of rats were detected by spectrophotometry to analyze the antioxidant effects of different low-dose insulin glargine interventions.

RESULTS: Compared with the sham group, the blood glucose of the rats in the delayed resuscitation control group was significantly increased, the heart and kidney functions were significantly reduced, the oxidation capacity was enhanced, and the antioxidant indicators were significantly reduced. After the intervention of insulin glargine, with the increase of insulin glargine dose, the blood glucose, myocardial enzyme and renal function indicators of rats showed a gradual downward trend, the oxidation indicators continued to decrease, and the antioxidant indicators showed a gradual upward trend. When the dose was 2.0 U×kg-1×d-1, the blood glucose, LDH, CK, CK-MB, α-HBDH, AST, BUN, SCr, XOD and MPO were significantly lower than those in the delayed resuscitation control group [blood glucose (mmol/L): 5.91±0.25 vs. 11.76±0.36, LDH (U/L): 3 332.12±51.61 vs. 5 008.94±490.12, CK (kU/L): 0.49±0.03 vs. 0.85±0.04, CK-MB (U/L): 125.40±12.19 vs. 267.52±11.63, α-HBDH (U/L): 122.99±5.37 vs. 240.85±13.99, AST (U/L): 11.95±1.81 vs. 17.87±1.57, BUN (mmol/L): 4.72±0.15 vs. 7.16±0.34, SCr (μmol/L): 87.11±6.51 vs. 137.50±11.36, XOD (U/g): 166.29±3.27 vs. 204.90±4.82 in heart tissue, 63.51±1.46 vs. 79.69±1.75 in kidney tissue, MPO (U/g): 1.05±0.02 vs. 1.55±0.06 in heart tissue, 1.04±0.04 vs. 1.87±0.01 in kidney tissue, all $P < 0.05$], and CuZn-SOD, CAT, GSH-Px and T-AOC were significantly higher than those in the delayed resuscitation control group [CuZn-SOD (kU/g): 82.95±2.69 vs. 56.52±2.26 in heart tissue, 94.50±2.73 vs. 62.02±1.66 in kidney tissue, CAT (U/g): 36.07±2.01 vs. 15.15±2.22 in heart tissue, 184.49±4.53 vs. 156.02±3.96 in kidney tissue, GSH-Px (kU/g): 231.93±8.03 vs. 179.48±3.15 in heart tissue, 239.63±7.30 vs. 172.20±2.09 in kidney tissue, T-AOC (kU/g): 4.85±0.23 vs. 2.71±0.11 in heart tissue, 5.51±0.08 vs. 3.50±0.07 in kidney tissue, all $P < 0.05$].

CONCLUSIONS: Different low-dose of insulin glargine (≤ 2.0 U×kg-1×d-1) could exert antioxidant protection on the heart and kidney of rats with delayed resuscitation after burns, with a dose-dependent manner.

PMID:36567555 | DOI:10.3760/cma.j.cn121430-20220627-00604

Categorías: Protección miocárdica

Nationwide cardiovascular risk categorization: applying the European Society of Cardiology (ESC) guidelines to the Swedish National Diabetes Register

Lun, 12/26/2022 - 11:00

Eur J Prev Cardiol. 2022 Dec 26:zwac308. doi: 10.1093/eurjpc/zwac308. Online ahead of print.

ABSTRACT

AIMS: The 2021 European Society of Cardiology (ESC) guidelines recommend that patients with type 2 diabetes (T2D) with a very high cardiovascular disease (CVD) risk receive cardiovascular (CV)-protective glucose-lowering medication (glucagon-like peptide-1 receptor agonists or sodium-glucose co-transporter-2 inhibitors). This analysis compared previous prescribing practices with the ESC recommendations.

METHODS AND RESULTS: Patients in the Swedish National Diabetes Register (NDR) with T2D, aged 18-90 years, not receiving CV-protective glucose-lowering medication in 2017 were identified, and the ESC criteria for very high CVD risk was applied. The composite outcome of major adverse CV events (MACE; defined as CV death, non-fatal stroke or non-fatal myocardial infarction) during 2017 was calculated and the number of MACE avoided with semaglutide, an example of a CV-protective glucose-lowering medication, was estimated for patients within a certain CV risk score.Of the 320,028 patients in the NDR with T2D who were not receiving CV-protective glucose-lowering medication, 129,512 patients had a very high CVD risk. Patients with a very high CVD risk had a high incidence of MACE (75.4 events/1000 person-years), which was higher in those with atherosclerotic CVD (ASCVD) with and without elevated glycated haemoglobin (>9%; 136.5 and 90.8 events/1000 person-years, respectively). If patients with a very high CVD risk, according to the ESC, and ASCVD received semaglutide, 803 MACE may have been avoided in 2017.

CONCLUSIONS: This analysis highlights differences between previous prescribing practices in Sweden and the 2021 ESC guidelines, and offers strategies to prioritize CV-protective glucose-lowering medication for patients who would benefit most.

PMID:36567502 | DOI:10.1093/eurjpc/zwac308

Categorías: Protección miocárdica

Coronary Artery Disease and its Vascular Associates in Patients with Chronic Non-surgical Hypoparathyroidism

Lun, 12/26/2022 - 11:00

Clin Endocrinol (Oxf). 2022 Dec 25. doi: 10.1111/cen.14872. Online ahead of print.

ABSTRACT

CONTEXT: Patients with chronic hypoparathyroidism (cHypoPT) are prone to intracranial-calcification, cataract, and nephrocalcinosis. In this study, we systematically investigated the possibility of increased coronary-artery calcification (CAC) and coronary-artery disease (CAD) in them.

DESIGN: Cross-sectional PATIENTS AND METHODS: 94 non-surgical cHypoPT (M:F=50:44; age=45±15years) with 18.6±9.3 years of illness were assessed. Those with dyspnea, angina, syncope, abnormal electrocardiogram, echocardiography, or significant CAC underwent coronary-angiography or myocardial-perfusion-stress imaging. Their lipid parameters and hsCRP were compared with age-matched healthy controls (Group A, n =101). The prevalence of CAC in cHypoPT was compared with that of subjects referred from cardiology-clinics (Group B, n=148, age=52±11 years).

RESULTS: One of 94 cHypoPT had known CAD. On screening, 17 cHypoPT required evaluation for CAD. Two of 17 had severe coronary-stenosis and 12 showed subclinical-CAD. CAC and aortic-valve calcification occurred in 21.5% and 11.8%. Clinical and subclinical CAD, CAC, and aortic-valve calcification in cHypoPT ≥50 years of age was 8.1%, 27.0%, 52.8%, and 27.8%, respectively. Frequency of age-adjusted CAC was comparable between cHypoPT and control-group B (30.2% vs. 30.7%, P=0.93). Elevated hsCRP was higher in cHypoPT than in controls-A (52% vs. 32%, P<0.01). Factors associated with CAD in cHypoPT were CAC and hypertension. However, CAD and CAC showed no association with long-term calcemic or phosphatemic control and intracranial-calcification in cHypoPT.

CONCLUSIONS: Clinical and subclinical CAD was observed in 3.2% and 12.8% of cHypoPT patients. The increased prevalence of CAD, CAC and aortic-valve calcification in cHypoPT above 50 years of age suggested their careful cardiac evaluation during follow-up.

PMID:36567495 | DOI:10.1111/cen.14872

Categorías: Protección miocárdica

UpStreAm doxycycline in ST-eLeVation myocArdial infarction - targetinG infarct hEaling and ModulatIon (SALVAGE-MI trial)

Lun, 12/26/2022 - 11:00

Eur Heart J Acute Cardiovasc Care. 2022 Dec 26:zuac161. doi: 10.1093/ehjacc/zuac161. Online ahead of print.

ABSTRACT

BACKGROUND AND AIMS: Experimental studies demonstrate protective effects of doxycycline on myocardial ischemia-reperfusion injury. The trial investigated whether doxycycline administered prior to reperfusion in patients presenting with ST-elevation myocardial infarction (STEMI) reduces infarct size (IS) and ameliorates adverse left ventricular (LV) remodeling.

METHODS: In this randomized, double-blind, placebo-controlled trial, patients presenting with STEMI undergoing primary percutaneous coronary intervention (PPCI) were randomized to either intravenous doxycycline or placebo prior to reperfusion followed by 7-days of oral doxycycline or placebo. The primary outcome was final IS adjusted for area-at-risk (fIS/AAR) measured on two cardiac magnetic resonance scans ~6 months apart.

RESULTS: Of 103 participants, 50 were randomized to doxycycline and 53 to placebo and were matched for age (59 ± 12 vs. 60 ± 10 years), male sex (92% vs. 79%), diabetes mellitus (26% vs. 11%) and left anterior descending artery occlusion (50% vs. 49%), all $p > 0.05$. Patients treated with doxycycline had a trend for larger fIS/AAR (0.79 [0.5-0.9] vs. 0.61 [0.47-0.76], $p = 0.06$), larger fIS at 6 months (18.8% [12-26] vs. 13.6% [11-21], $p = 0.08$), but similar acute IS (21.7% [17-34] vs. 19.4% [14-27], $p = 0.19$) and AAR (26% [20-36] vs. 24.7% [16-31], $p = 0.22$) compared to placebo. Doxycycline did not ameliorate adverse LV remodeling (%Δend-diastolic volume index, 1.1% [-3.8-8.4] vs. -1.34% [-6.1-5.8], $p = 0.42$) and was independently associated with larger fIS (regression coefficient = 0.175, $p = 0.03$).

CONCLUSION: Doxycycline prior to PPCI neither reduced IS acutely or at 6 months nor attenuated adverse LV remodeling. These data raise safety concerns regarding doxycycline use in STEMI for infarct modulation and healing.

PMID:36567466 | DOI:10.1093/ehjacc/zuac161

Categorías: Protección miocárdica

A Self-Sustaining Antioxidant Strategy for Effective Treatment of Myocardial Infarction

Dom, 12/25/2022 - 11:00

Adv Sci (Weinh). 2022 Dec 25:e2204999. doi: 10.1002/advs.202204999. Online ahead of print.

ABSTRACT

Myocardial infarction (MI) is the leading cause of death worldwide and can lead to the loss of cardiac function and heart failure. Reactive oxygen species (ROS) play a key role in the pathological progression of MI. The levels and effects of ROS are significantly different in three unique pathological stages of MI, and most antioxidants cannot make corresponding adjustments to eliminate ROS, which leads to a great compromise to treat MI with antioxidants. Herein, an innovative self-sustaining antioxidant strategy is developed to treat MI with self-sustaining selenium-embedded nanoparticles (SSSe NPs). SSSe NPs possess unique self-sustaining antioxidant effects at different pathological stages of MI. This strategy of on-demand ROS elimination during different pathological stages demonstrated excellent MI treatment efficacy and effectively reversed heart failure to normal heart function. The therapeutic mechanism of SSSe NPs is intensively investigated through a series of experiments and mainly involved five critical aspects of myocardial repair: protecting mitochondria, reducing cardiomyocyte apoptosis and ferroptosis, reducing inflammation and fibrosis, and promoting angiogenesis. This strategy not only provides a promising treatment option for MI but also offers inspiration for other ischemic diseases.

PMID:36567266 | DOI:10.1002/advs.202204999

Categorías: Protección miocárdica

Review of current ECG consumer electronics (pros and cons)

Dom, 12/25/2022 - 11:00

J Electrocardiol. 2022 Dec 17;77:23-28. doi: 10.1016/j.jelectrocard.2022.11.010. Online ahead of print.

ABSTRACT

BACKGROUND: Several wearable, medical-grade consumer ECG devices are now available and integrated into consumer electronics like multi sensor fitness watches and scales. Specific consumer ECGs can also come in the form of patches or thin sensor plates in credit card or other shapes. Watches with ECG capabilities are often multi vital sign sensor devices. The majority of these devices are usually connected to a mobile smartphone. However, there are pros and cons to their use.

METHODS: We review here an exemplary selection of modern consumer ECG devices based on device type, recording method and the number of standard ECG channels derived.

RESULTS: Single-channel consumer ECG devices such as Smart Watches can be useful for detecting and monitoring atrial fibrillation and flutter and other arrhythmias, as well as ectopic complexes. However, they are currently limited with respect to recording duration and information content (a single-channel or limb-lead ECG having less diagnostic information than a 12-lead ECG). While some non watch-based consumer ECG devices can now record all 6 limb leads to yield increased information, no consumer ECG devices can currently reliably detect ST-segment deviations, potentially indicating myocardial infarction or ischemic episodes. Moreover, barriers to use still exist for at-risk elderly people. Finally, there currently is no universal data exchange format.

CONCLUSION: Consumer ECG devices, whether in fitness or fashionable design, allow for reliable detection of atrial fibrillation. Timely detection of atrial fibrillation and subsequent treatment might protect against stroke, especially in high-risk groups, yet prospective evidence is still lacking. Six-channel consumer ECG and longer data collection capabilities extend potential functionality, including for the monitoring of ST-segments and QT intervals. However, no currently available devices are sufficiently suitable for the detection of myocardial infarction or ischemia, which is why portable 12-channel technologies are desirable. For the reliable detection of a myocardial infarction, the determination of specific myocardial infarction blood markers and evaluation of patient medical history still is indispensable in addition to the 12 lead ECG.

PMID:36566580 | DOI:10.1016/j.jelectrocard.2022.11.010

Categorías: Protección miocárdica

Improved precision of SPECT myocardial blood flow using a net tracer retention model

Sáb, 12/24/2022 - 11:00

Med Phys. 2022 Dec 24. doi: 10.1002/mp.16186. Online ahead of print.

ABSTRACT

BACKGROUND: Non-invasive quantification of absolute myocardial blood flow (MBF) and myocardial flow reserve (MFR) provides incremental benefit to relative myocardial perfusion imaging (MPI) to diagnose and manage heart disease. MBF can be measured with single-photon emission computed tomography (SPECT) but the uncertainty in the measured values is high. Standardization and optimization of protocols for SPECT MBF measurements will improve the consistency of this technique. One element of the processing protocol is the choice of kinetic model used to analyze the dynamic image series.

PURPOSE: This study evaluates if a net tracer retention model (RET) will provide a better fit to the acquired data and greater test-retest precision than a one-compartment model (1CM) for SPECT MBF, with (+MC) and without (-MC) manual motion correction.

METHODS: Data from previously acquired rest-stress MBF studies (31 SPECT-PET and 30 SPECT-SPECT) were reprocessed ±MC. Rate constants (K1) were extracted using 1CM and RET, +/-MC, and compared pairwise with standard PET MBF measurements using cross-validation to obtain calibration parameters for converting SPECT rate constants to MBF and to assess the goodness-of-fit of the calibration curves. Precision (coefficient of variation of test re-test relative differences, COV) of flow measurements was computed for 1CM and RET ±MC using data from the repeated SPECT MBF studies.

RESULTS: Both the RET model and MC improved the goodness-of-fit of the SPECT MBF calibration curves to PET. All models produced minimal bias compared with PET (mean bias < 0.6%). The SPECT-SPECT MBF COV significantly improved from 34% (1CM+MC) to 28% (RET+MC, p = 0.008).

CONCLUSION: The RET+MC model provides a better calibration of SPECT to PET and blood flow measurements with better precision than the 1CM, without loss of accuracy. This article is protected by copyright. All rights reserved.

PMID:36565461 | DOI:10.1002/mp.16186

Categorías: Protección miocárdica

Mitochondrial transplantation protects against sepsis-induced myocardial dysfunction by modulating mitochondrial biogenesis and fission/fusion and inflammatory response

Sáb, 12/24/2022 - 11:00

Mol Biol Rep. 2022 Dec 24. doi: 10.1007/s11033-022-08115-4. Online ahead of print.

ABSTRACT

BACKGROUND: Sepsis-induced myocardial dysfunction is associated with worse clinical outcomes and high mortality, but no effective therapeutic intervention has been explored, reinforcing the urgent need to develop innovative strategies. Mitochondrial dysfunction underlies the pathogenesis of sepsis-induced myocardial dysfunction. Herein, we assessed the effect of mitochondrial

transplantation on sepsis-induced myocardial dysfunction in a rat model of cecal ligation and puncture (CLP)-induced sepsis.

METHODS: Male Wistar rats (n = 80, 12 weeks old, 250-300 g) were divided into groups with/without CLP-induced sepsis receiving mitochondrial transplantation in single or two repetitive injections (1 h or 1 and 7 h post-CLP, respectively). Mitochondria were isolated from donor rats and injected intravenously (400 µl of mitochondrial suspension containing 7.5 × 106 mitochondria/ml of respiration buffer) in recipient groups. Twenty-four hours post-operation, LDH and cTn-I levels, mitochondrial functional endpoints, expression of mitochondrial biogenesis (SIRT-1 and PGC-1α) and fission/fusion (Drp1/Mfn1 and Mfn2) genes, and inflammatory cytokines (TNF-α, IL-1β, and IL-6) levels were evaluated. Survival was tested over 72 h post-operation.

RESULTS: Mitotherapy significantly improved 72-hours survival (P < .05) and decreased LDH and cTn-I levels (P < .01). It also restored mitochondrial function and expression of mitochondrial biogenesis and fusion genes, and decreased the expression of mitochondrial fission gene and the levels of inflammatory cytokines (P < .05 to P < .01). Mitotherapy with repetitive injections at 1 and 7 h post-CLP provided noticeable mitoprotection in comparison with the group receiving mitotherapy at single injection.

CONCLUSION: Mitotherapy improved mitochondrial function, biogenesis, and dynamic associated with SIRT-1/PGC-1α network and suppressed inflammatory response in CLP-induced sepsis model, therefore, offers a promising strategy to overcome life-threatening sepsis challenge.

PMID:36565415 | DOI:10.1007/s11033-022-08115-4

Categorías: Protección miocárdica

Efficacy and Safety of Calcium Channel Blockers in Preventing Cardiac Siderosis in Thalassemia Patients: An Updated Meta-Analysis with Trial Sequential Analysis

Sáb, 12/24/2022 - 11:00

Eur J Haematol. 2022 Dec 24. doi: 10.1111/ejh.13919. Online ahead of print.

ABSTRACT

OBJECTIVES: Iron overload in patients with thalassemia represents a serious complication by affecting numerous organ systems. This meta-analysis aims to establish an evidence regarding the effect of amlodipine on cardiac iron overload in thalassemia patients.

METHODS: We searched PubMed, Scopus, Web of Science, Cochrane Central, and EMBASE for all relevant randomized controlled trials (RCTs). The primary outcomes were cardiac T2* and myocardial iron concentration (MIC). Secondary outcomes were liver iron concentration (LIC), risk of G.I. upset and risk of lower limb edema. We used Hedges' g to pool continuous outcomes, while odds ratio was used for dichotomous outcomes.

RESULTS: Seven RCTs were eligible for this systematic review and meta-analysis, comprising of 233 patients included in the analysis. Amlodipine had a statistically significant lower MIC (Hedges' g = -0.82, 95% CI [-1.40, -0.24], p < 0.001) and higher cardiac T2* (Hedges' g = 0.36, 95% CI [0.10, 0.62], p = 0.03). Amlodipine was comparable to standard chelation therapy in terms of the risk of lower limb edema and GI upset.

CONCLUSION: Our meta-analysis found that amlodipine significantly increases cardiac T2* and decreases MIC, hence decreasing the incidence of cardiomyopathy-related iron overload in thalassemia patients.

PMID:36565288 | DOI:10.1111/ejh.13919

Categorías: Protección miocárdica

MiR-361-5p Inhibits the Wnt Axis via Targeting Lgr4 and Promotes Sepsis-induced Myocardial Injury

Vie, 12/23/2022 - 11:00

Ann Clin Lab Sci. 2022 Nov;52(6):927-937.

ABSTRACT

OBJECTIVE: A growing body of evidence demonstrated that microRNAs (miRNAs) play a key role in sepsis-induced organ dysfunction. However, the mechanism of miR-361-5p in sepsis-induced myocardial injury remains to be clarified.

METHODS: A mouse model of sepsis-induced myocardial injury was established using lipopolysaccharide (LPS). MiR-361-5p expression level was determined by quantitative reverse transcription-polymerase chain reaction (RT-qPCR). G protein-coupled receptor-4 (Lgr4), apoptosis-related proteins, and the Wnt signaling pathway-related proteins were determined by Western blotting. The relationship between miR-361-5p and Lgr4 was determined using dual-luciferase reporter (DLR) and RNA immunoprecipitation (RIP) assays.

RESULTS: MiR-361-5p expression level was upregulated in the mouse model of sepsis-induced myocardial injury, while an opposite result was found for Lgr4 expression level. Knockdown of miR-361-5p protected the mouse model of sepsis-induced myocardial injury against inflammation and oxidative stress, and reduced cardiomyocyte (CM) apoptosis, which could be reversed by knockdown of Lgr4. The analysis of underlying mechanism revealed that miR-361-5p could target Lgr4 to modulate the activity of Wnt axis in CM apoptosis.

CONCLUSION: MiR-361-5p could aggravate myocardial injury in LPS-induced septic mice by targeting Lgr4 to inhibit the Wnt axis.

PMID:36564072

Categorías: Protección miocárdica

- 1
- 2
- 3
- siguiente ›
- última »