

Cardiorenal Syndromes. New Drugs for Dysfunction of the Kidney and the Heart

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When dysfunction in one organ induce dysfunction in the other? In the American diabetes meeting 79th the complications of diabetes in chronic kidney disease and cardiovascular disease on microvascular and macrovascular complications is important Issue. CKD is a factor risk of CVD. We have a new evidence for credence and carmelina trials with the news drugs canagliflozin. The main results shown 30 per cents of reduction in the primary outcome of the end stage kidney disease, doubling of serum creatinine, death, from renal or cardiovascular causes. the reduction of cardiovascular death or hospitalization for heart failure were 26 per cent without cardiovascular disease, and 34 per cent with CVD. The risk of myocardial infarction, death, stroke was 32 and 15 per cents lower in both groups. CV benefits with diabetes medication was good and the renal outcome were identical. The carmelina trial with linagliptin dpp4 inhibitor results possible reversible kidney damage, 14 per cent of reduction of progression albuminuria and no cardiovascular benefits.

Dapagliflozin in the DAPA-CKD trial an international, multicenter, randomized, double blind, in 4245 patients with stage 2 - 4 chronic kidney disease. Patients receive 10 MGRS or a placebo. The primary endpoints is worsening of renal function, defined estimated glomerular filtration rate decline 50 per cent and death for cardiovascular or renal causes. Dapagliflozin is a sodium glucose transporter 2 inhibitor currently indicated for treatment of diabetes type 2 and for reduction of the risk of hospitalization for heart failure. The 5 year risk of death in patients who develop heart failure with type 2 diabetes mellitus is 48 per cent of 3 times higher that patients who are not heart failure or comorbidities. The most common comorbidities is heart problem 8 per cent, stroke 3 per cent, CKD 2.2 per cent occurred after 5 years and 4 per cent after 10 years. The most deadly comorbidities heart failure, second stroke and pad, and CKD. Heart failure and CKD with PVD, each of the combinations boosted mortality by 300 to 400 per cent when occurred during the first year after T2 D diagnosis. The ENPAREG- trial outcome had a solid reduce need to either start insulin treatment or intensify existing insulin therapy, compare with placebo. EMPAREG outcome trial shown substantial decrease in heart failure hospitalization, incident of heart failure and progression of renal dysfunction. Lixisenatide in patients with type 2 diabetes who have cardiovascular disease and macroalbuminuria may have improve in ELIXA trial. Dulaglutide show significant reduce cardiovascular events in rewinds trial. There is a few new drugs for cardiac failure the inotropes non depending of calcium metabolism like Omecantiv mecarbil is a activator of the myosin, vericiguat is an stimulator of GMP, and the sacubitril neprilysin offer a new target for therapeutics neprilysin is an enzyme that degrade endogenous natriuretic peptides, these peptides are secreted during time fluids overload and the effect of neprilysin is improve the natriuretic effect. The combination with valsartan in the paradigma heart failure trial shown improves of cardiovascular outcome, the hypothesis that dual therapy preserve the kidney better than monotherapy former the basis UK Harp-3 trial , but the trial was negative in cardiovascular outcome, and kidney. Ultrafiltration for heart failure with cardiorenal syndrome in the CARRESS trial was disappointing. The pathophysiological mecha-

nism in cardiorenal syndrome in CKD include neurohumoral, hemodynamic and related CKD. The sympathetic nerve activity play a role in this syndrome but renal denervation simpliciter fail after catheter denervation. We news to know if this drug are useful and safety in cardiorenal syndrome. we need a large randomized trial to see the results in mortalities and qualities of life. Fibroblast growth factor 23 is phosphaturic hormone has recently identify in CKD related factor in cardiorenal syndrome. Higher serum phosphorus level and lower glomerular filtration stimulate upregulation of fibroblast growth factor which stimulate the excretion of phosphorus. Lowering serum phosphorus and fgh23 has significant cardiovascular effect with phosphate binder and inhibitor fgh23 in cardiorenal syndrome [1-6].

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