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Global Summit on NEPHROLOGY, UROLOGY AND KIDNEY TRANSPLANTATION

June 15, 2022 | Webinar

Pathophysiological aspects of dyspepsia in patients with chronic kidney disease undergoing hemodialysis

Olgica Mihaljevic[1], Batric Babovic[2]

[1]*University of Kragujevac, Faculty of Medical sciences, Department of pathophysiology, Serbia*

[2]*Clinic for Nephrology, Clinal Centre of Montenegro, Podgorica, Montenegro*

Chronic kidney disease (CKD) is associated with a number of complications, including dyspepsia. It is estimated that about 80% of patients with CKD have some gastrointestinal disturbances. Dyspepsia refers to the occasional or constant epigastric pain, and clinically it is presented as: 1) postprandial distress syndrome (postprandial fullness, early satiety, bloating, nausea and excessive belching) and/or 2) epigastric pain syndrome. It could be organic (associated with certain diseases) or functional dyspepsia (the absence of any organic, systemic or metabolic disease). Pathophysiological aspects of dyspepsia in CKD patients undergoing hemodialysis have been insufficiently studied. It is considered that uremic toxins and uremic neuropathy, digestive hypomotility, visceral hypersensitivity to gastric distension, amyloid protein deposition and Helicobacter pylori (HP) infection have important role in the development of dyspeptic symptoms in dialysis population. As we demonstrated in our previosly conducted investigations, CKD patients undergoing hemodialysis commonly presented dyspepsia as occasional epigastric pain, bloating and nausea. The most common pathohistological finding was chronic atrophic gastritis which positively correlated with dialysis duration in our study population. The estimated prevalence of HP in CKD patients undergoing hemodialysis was over 50 percent. HP presence was considerably greater in the patients underwent hemodialysis for a shorter time. Also, HP was more common in the subjects with higher values of bicarbonate. It seems that HP induces certain histopathological changes by modulation of gastric secretion and dysfunction of gastric smooth muscles during host inflammatory responce.