

GASTROINTESTINAL(GI) MANIFESTATIONS OF PATIENTS WITH CHRONIC KIDNEY DISEASE

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ABSTRACT: Chronic Kidney Disease (CKD) is a serious public health problem worldwide. One of the most common disorders in patients with CKD is also gastrointestinal disorders (GI). GI diseases are one of the most obvious manifestations of the disorder between hemostatic balance, hemodynamic and immunological balance in patients with CKD. Gastrointestinal symptoms are manifested in the early stages of the disease. There are currently no clear guidelines for managing the many gastrointestinal problems that occur in patients with CKD. **Purpose of the paper:** The paper aims to verify the presentation, diagnosis, progress and the prognosis of gastrointestinal disorders (GI) according to the role of different segments of the digestive system in patients with chronic kidney disease. This paper consists of describing the specific phenomena of gastrointestinal diseases present in patients with CKD. A large number of studies have verified that there is a high positive correlation between GI and CKD diseases and the chronic damage of the kidneys itself significantly affects the appearance of disorders and lesions in the gastrointestinal tract. This paper reviews existing data on gastrointestinal complications in patients with CKD as well as the management of the most common GI disorders that occur in patients with CKD. **Conclusion:** Since gastrointestinal complaints in patients with chronic kidney disease appear in the early stages of the disease in conclusion we may prefer that their treatment and management should begin as soon as possible, that in the initial stages of the appearance of GI symptoms. Because the pathogenesis of GI disorders in patients with CKD is relatively more complex in which are combined a number of causes is more than necessary to the earlier of the laboratory examination, bacteriological, endoscopic to detect etiology and the factor that leads to disturbances GI in order to avoid or treat as soon as possible the factors that contribute to the appearance of symptoms. Therapies with proton pump inhibitors have shown good clinical efficacy not only in relieving gastrointestinal distress but also in improving renal function.

Keywords: gastrointestinal (GI) disorders Chronic kidney disease (CKD) ;

ENTRY

Gastrointestinal disorders (GI) are common in patients with chronic kidney disease (CKD), resulting in a lack of knowledge of the mechanisms leading to a wide range of symptoms, from nausea to anorexia and disturbances in sodium and potassium balance, malnutrition as well as the side effects of the therapy they use. It is estimated that about 80% of patients with CKD present with symptoms of gastrointestinal tract (GIT) disorders during the course of their disease, although a number of them have a prevalence of indigestion similar to individuals of the general population who have no chronic renal disease (1,2,3). However, some of them, such as indigestion, may have a prevalence not different from that of the general population. GIT symptoms in

patients with chronic kidney disease, has a multifactorial origin. As factors to frequent affecting GI counted cumulation toxic products (endogenous and exogenous), a change in the extracellular environment homeostasis, iatrogenic origin, the continuous inflammation trans mural of the intestinal mucosa, influence the underlying disease which leads to CKD etc.

PURPOSE OF THE PAPER: The paper aims to verify our discussion on the presentation, diagnosis, prognosis and prognosis of gastrointestinal disorders (GI) in patients with chronic kidney disease.

DISCUSSION

Patients with chronic kidney disease (CKD) typically experience gastrointestinal disorders, including dysgeusia, anorexia, indigestion, hiccups, nausea, and vomiting. Gastrointestinal hemorrhage occurs frequently and

may originate from peptic ulcer disease, vascular ectasia, amyloidosis, or diverticulosis, with uremic bleeding diathesis, ulcerogenic medications, and infections that contribute to this increased bleeding tendency. Peptic ulcer

disease, is spread similar to that of the general population, with two associated factors: helicobacteriozön and consumption of GI disorders NSAIDs. Menaxhimi ynon prevent or mitigate the emergence of systemic clinical manifestations resulting from reduced renal function and allow symptomatic management of patients with CKD. Common clinical manifestations in patients with CKD are: gingivitis, stomatitis, vomiting, nausea, esophagitis, gastro-oesophageal reflux, gastritis (acute and chronic) duodenitis, peptic ulcer, ex ureteric reflux ore, flatulence, ischemic colitis, etc. The treatment of these patients is no different from that given to the general population with proton pump inhibitors, drugs such as prokinetics: metoclopramide and domperidone. after endoscopic examination. Helicobacter pylori is associated with significant urease activity, with no statistically significant differences compared to patients on dialysis. In addition to conventional treatment of infection, strict diet control is required, especially with low phosphorus. In patients with CKD, levels of gastrin, cholecystokinin, and secretion are increased by increased secretion directly related to the degree of renal failure (4,5,6) .It has been reported that gastro-intestinal symptoms may be evident in patients with CKD at relatively high levels. high eGFR, long before ESRD, and will become increasingly evident as the disease progresses (7). Gastrointestinal disorders can not only affect the quality of life of patients with MRS, but also result in dehydration, electrolyte imbalance, malnutrition, which will further lead to kidney damage (8). The pathogenesis of gastroin-testinal disorders in MRS is relatively complex, more so than the role of uremic toxin. However, research on gastrointestinal disorders in CKD is still limited. Pathogenesis of pastro-intestinal disorders in patients with CKD. Various factors are involved in the development of gastrointestinal disorders, including accumulation of uremic toxins and increased gastrointestinal hormones with decreased renal function and changes in intestinal flora. All of these factors can lead to damage or destruction of the gastric mucosa barrier, or gastrointestinal motility, which will eventually result in disorders or gastrointestinal lesions. Uremic toxins, such as indoxyl sulfate and hippuric acid resulting in decreased function of the kidney, are important factors causing gastrointestinal damage. On the one hand, patients with renal failure have more urea excretion from the gastrointestinal tract, resulting in a significant increase in ammonia and carbonate that are broken down by bacteria, which will result in extensive inflammation and erosion of the mucosa. On the other hand, studies have shown that the cacu-mulation of uremic toxin may be the cause of gastrointestinal motor dysfunction, exhibiting increased transit time and impaired motility, and may further impair intestinal smooth muscle

contractility . Furthe-rmore, increasing the residence time of feces in the colon can further increase the production / absorption of uremic toxins derived from the colon and alter the reabsorption of water and electrolytes. Thus develops a vicious cycle of uremia in the colon (9,10). Activity of gastrointestinal hormones: a number of gastrointestinal hormones are excreted by the kidneys, so with the decline in kidney function, many gastrointestinal hormones such as: gastrin, motilin, cholecystokinin and vaso-intestinalshow increased circulating levels in patients with CKD. Altered levels of gastrointestinal hormone circulation can have a strong impact on intestinal motility. Motilin is an important gastroin-testinal hormone that primarily stimulates mechanical mobility in the gastrointestinal tract and regulates the rate of contraction of the gastrointestinal smooth muscle as well as the gallbladder. Accumulation of motilin in patients with SKD will cause disorders of gastrointestinal motility and abnormal secretion of gastric acid, leading to lesions such as ulcers. Gastrin has a motile-like function, and can elicit a contractile response of the antral gastric smooth muscle by combining with specific smooth muscle cell receptors (11,12,13). Because intestinal motility is regulated by the sympathetic system and parasympathetic systems, which inhibit and stimulate intestinal motility since in patients with CKD the activity of this system means increased activity of the sympathetic nervous system and depressive parasympathetic activity which will contribute to gastrointestinal dysmotility and delayed transit time. Autonomic nervous system dysfunction can be caused by uremic toxins which can directly damage small nerve fibers, and there are several other factors as well, such as activation of the renin-angiotensin-aldosterone system. Results from studies that have attempted to determine the association between renal dysfunction and pastroi-nestinal complications are limited by the small number of patients examined as well as by the various samples. There are currently no clear guidelines on managing the many gastrointestinal problems that correspond to CKD. A large number of studies have reported that gastrointestinal symptoms in patients with CKD are highly positively correlated with gastrointestinal motor dysfunctions and that the time of gastric transit will be prolonged and gastric myoelectric activity will change in patients with CKD (14). The most recent studies on GI disorders in patients with CKD have shown that changes in the intestinal flora, use of aspirin, non-rheumatic nonsteroidal anti-inflammatory drugs (NSAIDs) significantly affect the onset of GI symptoms. with CKD and can affect and accelerate renal damage it is preferable that gastrointestinal exami-nations begin in the early stages of the disease therefore their management with proton pump inhibitors(Nexium, Pantoprazole, Omeprazole, Lantoprazole,

etc.), domperidon not only has a significant advantage in alleviating gastrointestinal symptoms, but may also affect the

slowing of the progression and progression of chronic renal disease. (15).

CONCLUSION

Since gastrointestinal complaints in patients with chronic kidney disease appear in the early stages of the disease in conclusion we can prefer that their treatment and management should begin as soon as possible in the initial stages of GI symptoms. Because the pathogenesis of GI disorders in patients with CKD is relatively more complex in which are combined a number of causes is more than necessary to the earlier of the laboratory examination, bacteriologic,

endoscopic to detect etiology and the factor that leads to disturbances GI in order to avoid or treat as soon as possible the factors that contribute to the appearance of symptoms. Therapy with proton pump inhibitors has shown good clinical efficacy not only in relieving gastrointestinal distress, but also in improving renal function.

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