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MODERN SCIENTIFIC APPROACHES AND EFFECTIVE CORRECTION OF THE TREATMENT OF CHRONIC KIDNEY DISEASE IN PATIENTS WITH EROSIVE AND ULCERATIVE LESIONS OF THE STOMACH AND DUODENUM

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In the abstract there are studied pathogenetic peculiarities of appearance erosive damaging of stomach in patients with chronic kidney disease of IInd and IIIrd stage, which is caused by prolonged course of chronic pyelonephritis by the mean of investigation of serum proteolic actitvity of blood. There was investigated the state of unlimited proteolysis by the way of definition of lysis of asoalbumin (dissimilation lowmolecular proteins), asokasein (degradation of highmolecular proteins), and asokol (lysis of callogen). The appearance of erosive damaging of stomach in patients with chronic kidney failure of II and III stages with chronic pyelonphritis is revealed by elevated lysis of low- and highmolecular proteins and reliable elevation of collagenolytic activity of blood.

Keywords: chronic pyelonephritis, chronic kidney disease, asoalbumin,asokasein, asokol.

Statement of the problem. This paper presents main pathological changes of gastric mucosa in patients with chronic kidney disease II-III stages. Found that the most pronounced changes observed in patients with peptic ulcer combined with chronic kidney disease. Authors present the current state of the problem on gastric mucosal lesions in patients with chronic kidney disease.

We thoroughly studied the morphological changes of gastric mucosa at ultra structural level of gastric ulcer, gastritis with different etiology [1, 2, 3, 4, 15, 17]. However, in the literature there are works devoted to the study of lifetime structural changes in human gastric mucosa with underlying renal disease. Not studied the morphological changes of gastric mucosa in patients with chronic kidney disease (CKD).

Analysis of recent researches and publications. The problem is Helicobacter pylori's diseases which remains valid as because of their high frequency directly, and due to the very high infection – more than 80% of the adult population in Ukraine [5]. We know that from 7 to 11.5% of the population suffers from a combination of these pathologies [6, 14, 16, 19].

Histological examination of the stomach in patients with inflammatory lesions generally accounts for semi-quantitative assessment of severity of active and chronic inflammation, atrophy severity, including recognition scheme which came into their evaluation by MF Dixon (1994). However, working with remote tracking system the dynamics of these processes came after Hp eradication in recent years [1]. Only a single subject of study was the work of a homogeneous group of patients with duodenal ul-

cers [7]. Poorly understood and the pace of change are the characteristics of inflammatory factors that affect their dynamics, communication epithelialization of ulcers and erosions of gastric mucosa [8, 13, 14,]. Unquestionable impact on the damage of stomach is Helicobacter pylori (HP) [4, 18, 19].

The purpose of the work. These conditions encourage the depth of histo-pathological study of the dynamics as diffuse and focal processes in gastro duodenal CO in the treatment of peptic ulcer (SU) in combination with CKD II-III levels and assess their value as potential predictors of pathological processes in the course of the gastric mucosa [5].

Formulation purposes of article (problem). In the process of vital activity the human organism is constantly interacting with a host of microorganisms, resulting in a permanent selection of those strains that could colonize the mucous membranes (e.g., of the gastrointestinal tract or the urinary system), using it as a habitat medium. As a result of this selection a symbiosis is formed between micro-and macro-organisms, representing the normal microflora of the human body [1, 2].

The main material of the research. The aim of our study was to investigate morphological features of changes of gastric mucosa and their pathogenetic study at differentiated treatment in patients with chronic kidney disease stages II and III.

The allocation of unsolved aspects of the problem. Morphological studies were performed on biopsy of gastric mucosa that were selected during endoscopic study of patients, biopsy of the stomach was conducted in the morning on an empty stomach.

The study involved 78 patients, among them dominated by women – 58 (65.51%); there were 20 (34.48%). The average age of patients was 47,3 ± 2,6 years. The average duration of disease ranged from 7,2 ± 1,41 years, including 19 patients with CKD second degree with erosive ulcerous lesions of the stomach (EULS) in presence of H. pylori (group 1), 20 patients with CKD second degree with duodenum ulcer (DU) with the presence of H. pylori (2nd group), 21 patients with CKD third degree with EULS and without H. pylori (3rd group), 18 patients with CKD third degree with DU but without H. pylori (group 4). Age groups varied from 19 to 67. All figures are presented as mean values from their mean error (M ± m). The reliable difference is believed to be at p<0,05. Also conducted microscopic studies, based on the results of computer spectrometry according to the method of staining of histological sections. Statistical analysis of the results of research was conducted on the PC III using «Statistica 6,0».

The impressions of endoscopic signs of the gastric mucosa biopsies were histologically examinationed, which confirmed the presence of inflammatory changes of varying severity in the gastric mucosa.

Depending on the presence and severity of inflammation the gastric mucosa was detected in 19 patients EULS, Eve gastric mucosa – in 20 patients, DU – in 21 patients, and duodenum EULS – in 18 patients. EULS is characterized by preservation of architectonics gastric mucosa, while in the scale of glands: the principal, and additional parietal cells were detected in normal proportions, about 19, 38, 46 and 27% respectively of the total number of cells in the gland.

H. pylori averaged 84,2%, indicating preservation of functional activity of the gastric mucosa. Weak histological detection of inflammation in the stomach averaged 24.41% and was significantly (P<0,05). In the case of the studied patients H. pylori in the gastric mucosa with moderate to fundic parts that were on average 14,74% and were significantly (p <0,001) lower in comparison with those of at DU.

The development of the inflammatory reaction of the gastric mucosa with H. pylori, which averaged 47% and was significantly (p <0.05) lower as compared with those of patients with duodenal ulcer.

Thus, the comparison of data on the basis of path morphological study of biopsies and results regarding analysis of H. pylori and EULS showed significant reduction in the functional activity of the gastric glands in the development and progression of inflammation. Pathological studies revealed weak degree of inflammatory reaction in the stomach, which averaged 35% and was significantly (p <0,001) lower in comparison with those of at DU.

Thus, the comparison of data on the basis of path morphological studies in patients with CKD stage II with the presence of H. pylori EULS is likely to decrease in functional activity of the gastric mucosa, which depended on the progression of inflammation in it. In all cases path morphological examination accompanied by more or less marked changes in the gastric mucosa. Dysplasia was detected in areas of proliferative compartment of gastric glands containing cervical mukocyties.

The findings from this study. In patients with CKD stage III and concomitant presence of H. pylori and EULS found a significant positive correlation than in patients with CKD stage II and concomitant EULS without the presence of H. pylori between the severity of ulcerative process in GM and helikobakterioza level (r = + 0,712, p<0,001).

Compared with patients having CKD stage III of DU without H. pylori patients with CKD and second degree from DU with the presence of H. pylori this correlation was more moderate and was (r = + 0,417, p<0,05).

Analysis of the data indicates that patients with different CKD groups with and without the presence of H. pylori is connected with EULS, there is a significant change in the performance of different groups.

References:

1. Авраменко А.А. Хеликобактериоз. – Одесса: Фотосинтетика, // А.А. Авраменко, А.И. Гоженко // 2006. – 326 с.
2. Бардах Л.Б. Роль Helicobacter Pylori в патогенезі гастродуоденальних ерозій / Л.Б. Бардах // Сучас. інфекції. – 2005. – № 2. – С. 92-96.

3. Барышникова Н.В. Роль генетическх особенностей Helicobacter pylori в патогенезе заболеваний органов пищеварения: от теории к практике / Н.В. Барышникова, А.Н. Суворова, Е.И. Ткаченко, Ю.П. Успенский // Експерим. та клін. гастроэнтерол. – 2009. – № 1. – С. 12-19.
4. Бондаренко Т.В. Особливості морфологічного стану слизової оболонки шлунка у хворих на виразкову хворобу дванадцятипалої кишки залежно від наявності патогенності H.pylori / Т.В. Бондаренко // Сучасн. гастроентерол. – 2005. – № 4. – С. 41-45.
5. Зак М.Ю. Клініко-функціональні та морфологічні особливості виразкової хвороби дванадцятипалої кишки і хронічного гастриту у хворих на гіпертонічну хворобу та їх лікування: автореф. дис. на здобуття наук. ступеня канд. мед. наук / М.Ю. Зак. // – Дніпропетровськ, 2005. – 23 с.
6. Кашин С.В. Атрофия, метаплазия, дисплазия – факторы риска развития рака желудка: обратимы ли эти изменения слизистой оболочки? / СВ. Кашин, А.С. Надеждин, И.О. Иванников // Клин.перспективы гастроэтерол., гепатол. – 2007. – № 2. – С. 13-17.
7. Козлов В.В. Функционально-морфологические особенности слизистой оболочки желудка у больных язвенной болезнью двенадцатиперстной кишки молодого возраста при лечении ингибиторами протонной помпы: автореф. дис. на соискание учен. степени канд. мед. наук / В.В. Козлов // – СПб, 2006. – 21 с.
8. Мойсеєнко В.О. Гастроентерологічні розлади при вторинних нефропатіях // В.О. Мойсеєнко Актуальні проблеми нефрології (Вип. 6) // За ред. Т.Д. Никули. – К.: Задруга, 2006. – С. 236-238.
9. Lopez T., Quesada M., Almirall J., Sanfeliu I., Segura F., Calvet X. Usefulness of non-invasive tests for diagnosing Helicobacter pylori infection in patients undergoing dialysis for chronic renal failure // Helicobacter. – 2004. – Vol. 9, № 6. – P. 674-680.
10. Lorette C. Immunocytochemical methods and protocols. – Javois.: Humana Press, 2000. – 233 p.
11. Mak S.K., Loo C.K., Wong P.N., Lo K.Y., Tong G.M., Lam E.K., Wong A.K. A retrospective study on efficacy of proton-pump inhibitor-based triple therapy for eradication of Helicobacter pylori in patients with chronic renal failure // Singapore Med. J. – 2003. – Vol. 44, № 2. – P. 74-78.
12. Mehrotra R., Nolph K.D. Treatment of advanced renal failure: low-protein diets or timely initiation of dialysis? // Kidney Int. – 2000. – № 58 (4). – P. 1381-1388.
13. Nakahama H., Yanaka Y. Elevated serum pepsinogens in chronic renal failure patients // Nephron. – 1995. – V. 70, № 2. – P. 211-216.
14. Nardone G., Rocco A., Fiorillo M. ET al. Gastroduodenal lesions and Helicobacter pylori infection in dyspeptic patients with and without chronic renal failure // Helicobacter. – 2005. – Vol. 10, № 1. – P. 53-58.
15. Stadtman E.R., Berlett B.S. Reactive oxygen-mediated protein oxidation in aging and disease // Drug. Metab. Rev. – 1998. – Vol. 30, № 2. – P. 225-243.
16. Stadtman E.R., Levine R.L. Protein oxidation // Ann. N. Y. Acad. Sci. – 2000. – Vol. 899. – P. 191-208.
17. Tannert C., Lux W. Spreding of red blood cell suspensions on paper as simpel test of cell deformability // Acta biol. med. germ. – 1981. – Vol. 40, № 6. – P. 739-742.
18. Uchida K., Kanematsu M., Morimitsu Y. et al. Acrolein is a product of lipid peroxidation reaction. Formation of free acrolein and its conjugate with lysine residues in oxidized low density lipoproteins // J. Biol. Chem. – 1998. – Vol. 273, № 26. – P. 16058-16066.
19. Yao Zhengbin, Painter Sally L., Fanslow William C. et al. Human IL-17; A novel cytokine derived from T cell // J. Immunol. -1995. – Vol. 115. – № 12. – P. 5483-5486.

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СУЧАСНІ НАУКОВІ ПІДХОДИ І ЕФЕКТИВНА КОРЕКЦІЯ ЛІКУВАННЯ ХРОНІЧНОГО ЗАХВОРЮВАННЯ НИРОК У ХВОРИХ З ЕРОЗИВНО-ВИРАЗКОВИМ УРАЖЕННЯМ ШЛУНКА ТА ДВАНАДЦЯТИПАЛОЇ КИШКИ

Анотація
В роботі вивчено деякі патогенетичні особливості виникнення виразкових уражень шлунка у хворих на хронічну хворобу нирок II і III ступеня, обумовлену тривалим перебігом хронічного рецидивуючого пієлонефриту на підставі дослідження протеолітическої активності плазми крові. Досліджено стан необмеженого протеолізу шляхом визначення лізису азоальбуміну (розпад низькомолекулярних білків), азоказеіну (деградація вісокомолекулярніх білків) і азоколу (лізис колагену). Виникнення виразкових уражень шлунка у хворих на хронічну хворобу нирок II і III ступеня з наявністю хронічного пієлонефриту супроводжується істотним збільшенням лізису низько- і високомолекулярних білків і ймовірним зростанням колагенолітичної активності крові.
Ключові слова: хронічний пієлонефрит, хронічна хвороба.

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СОВРЕМЕННЫЕ НАУЧНЫЕ ПОДХОДЫ И ЭФФЕКТИВНАЯ КОРРЕКЦИЯ ЛЕЧЕНИЯ ХРОНИЧЕСКОГО ЗАБОЛЕВАНИЯ ПОЧЕК У БОЛЬНЫХ С ЭРОЗИВНО-ЯЗВЕННЫХ ПОРАЖЕНИЙ ЖЕЛУДКА И ДВЕНАДЦАТИПЕРСТНОЙ КИШКИ

Аннотация
В работе изучено некоторые патогенетические особенности возникновения язвенных поражений желудка у больных хронической болезнью почек II и III степени, обусловленную длительным течением хронического рецидивирующего пиелонефрита на основании исследования протеолитическои активности плазмы крови. Исследовано состояние неограниченного протеолиза путем определения лизиса азоальбумину (распад низкомолекулярных белков), азоказеину (деградация високомолекулярних белков) и азоколу (лизис коллагена). Возникновение язвенных поражений желудка у больных хронической болезнью почек II и III степени с наличием хронического пиелонефриту сопровождается существенным увеличением лизиса низко- и высокомолекулярных белков и вероятным ростом колагенолитичнои активности крови.
Ключевые слова: хронический пиелонефрит, хроническая болезнь