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МОРФОЛОГИЯ ЖЕЛУДОЧНОЙ СЛИЗИ ПРИ НЕКОТОРЫХ КЛИНИЧЕСКИХ ВАРИАНТАХ ЯЗВЕННОЙ БОЛЕЗНИ ЖЕЛУДКА

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MORPHOLOGY OF GASTRIC MUCOSA AT SAME CLINICAL VARIANTS OF ULCER DISEASE

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Аннотация

На экспериментальном материале (желудочная слизь, гомогенат слизистой оболочки желудка), полученном при фиброгастродуоденоскопии у 12 практически здоровых людей и 30 пациентов с язвенной болезнью, в том числе осложненной перфорацией, кровотечением или пенетрацией (12), впервые показано, что наличие соконтаминации слизистой оболочки желудка Helicobacter pylori и микроорганизмами родов Providencia и Morganella сочетается с высокими кристаллогенными свойствами данной среды, что способно провоцировать развитие осложнений язвенной болезни желудка.

Ключевые слова: патогенный симбиоз, микроорганизм-ассоциированный кристаллогенез, язвенная болезнь желудка

Abstract

We investigated crystallogenic and initiated properties of gastric mucosa and gastric mucosal homogenates in 12 healthy peoples and 30 patients with ulcer disease (12 patients also have bleeding or perforation or penetration). It is stated, that cocontamination of Helicobacter pylori and Providencia or Morganella combinates with high crystallogenic properties of biological fluids. It may be useful for ulcer disease pathogenesis investigation.

Keywords: pathogenic symbiosis, microorganism-associated crystallogenesis, ulcer disease.

Microogranism possibility to crystallogenesis initiation was stated long ago. So, it was shown by V.F. Chubukov (1982), that bacteria can formed many variants of crystal and pseudocrystal structures [4]. These data was confirmated by other investigators [1, 5]. We named illustrated phenomenon as «microorganism-associated crystallogenesis» (MAK). On our opinion, it has number of function, such as protective, pathogenic etc [2]. Literature data analysis shown, that in natural conditions protective function of MAK is dominanted, and pathogenic function realize at bacterial antagonism or infection process [1, 2].

Special variant of MAK are a bacterial symbiosis with high crystallogenic activity. Example of this symbiosis is microbial induced film-formation in catheters.

Aim of this paper is estimation of crystallogenic properties of gastric mucosa in connection with its microbial contamination.

Material and methods. We investigated crystallogenic properties of number of biological substrata (gastric mucosa, gastric mucosal layer homogenates) of 12 healthy peoples and 30 patients with ulcer disease, including complicated by perforation, bleeding or penetration (12 patients). Biological substrata were gotten at fibrogastroduodenoscopy.

Estimation of crystallogenic and initiated properties of biological material was accomplished by own methods (classic crystalloscopy and comparative teziography [2]). We used 0,9% sodium chloride solution as basic substance in teziographic test.

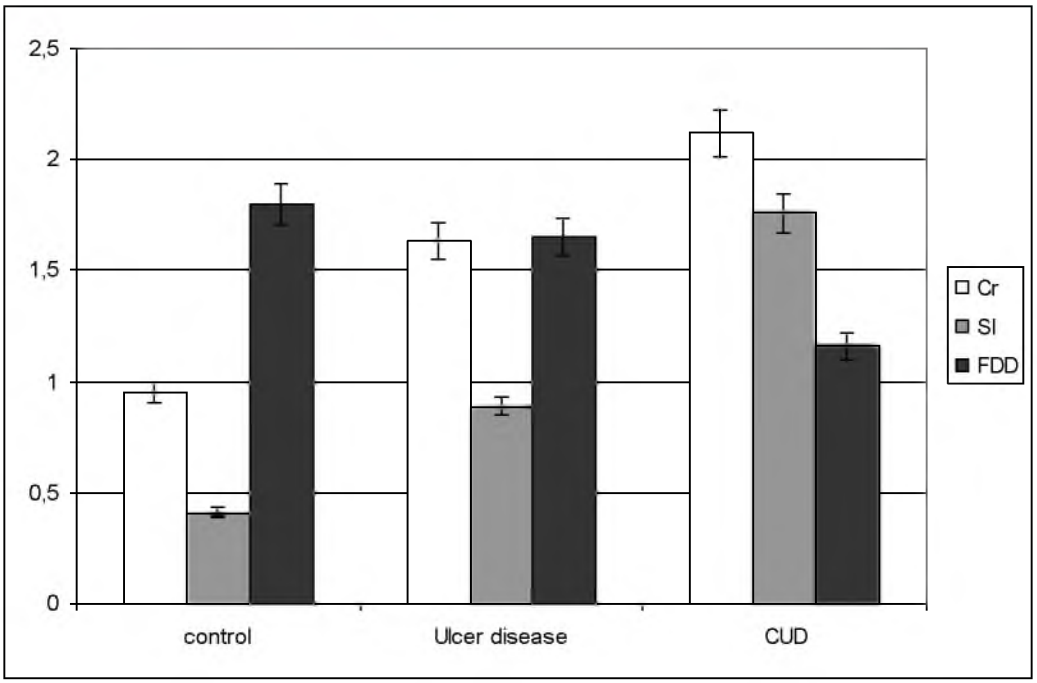

Fig. 1. Visuometric estimation of crystllogenic properties of gastric mucosa (Cr – crystallizability, SI – structure index, FDD – facia destruction degree, CUD – complicated ulcer disease)

Biological substrata crystalloscopic investigation was accompanied by its traditional microbiological study for Helicobacter pylori (Hp) or/and other microorganism's detection.

Results. Investigation of own and initiated crystallogenesis of gastric mucosa in peoples without gastrointestinal pathology allows to state in this case biological material has low crystallogenic activity. At dehydration it mainly formed numerous amorphic structures and single small crystals. Described crystalloscopic picture of gastric mucosa connected with no Helicobacter pylori detection in biological substrate. So, in physiological conditions bacterial flora and gastric mucosa, contained bulk mucopolysaccharides [3], have no essential crystallogenic potential and do not promote material initiation of basic substance crystallogenesis in teziographic test (fig. 1).

At endoscopic evidence of stomach ulcer defect presence, induced by Hp infection, crystallizability and initiation potential of gastric mucosa are meaningfully higher, than at healthy people ($p<0,05$). It manifested as elevation of crystalloid structures part in facia, caused crystallizability rate augmentation, but this tendency does not accompanied formed elements complication (structure index saving at normal level; $p>0,05$). These changes were corroborated by morphological and morphometric analysis of gastric mucosal layer homogenates dehydrated samples. In its facias only single small crystals are presented.

At complicated variants of ulcer disease marked crystallogenesis activation in investigated biological fluids is visualized. So, in this case crystallograms of gastric mucosa and gastric mucosal layer homogenates include numerous single-crystal elements and dendrites. It caused elevation of crystallizability and structure index level in comparison with healthy people and patients with non-complicatied ulcer disease rate. It is interesting, that forming crystals have rather low degree of destruction ($1,42\pm0,31$ rel. un.), which can indicate its importance in this pathogenic type of ulcer disease. This tendency visualized in teziographic facias too. At morphological analysis of gastric mucosal layer homogenates its high crystallogenic properties were discovered. Crystallograms of this biological substrate contained dendrites. It is very important, that at microbiological study in all biological materials Providencia or Morganella bacteria were marked out in addition to Hp. On this base we conjecture, that its sympiosis can be an initial factor of gastric mucosa damage.

Conclusion. Our data allow to suppose cocontamination of stomach mucosa by Helicobacter pylori and Providencia or Morganella caused elevation of gastric mucosa crystallogenic properties, that provoked formation of ulcer. Procrystallogenic potential of this symbiosis may be an important link of ulcer disease pathogenesis, which realized throw microorganism-associated mucosa damage (result of MAK activation) and disease complications progress.

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