



impact of nonrenal determinants such as sex, race, age, constitution, and medications. Under condition of an acute drop in GFR, Scr is insensitive to small decrements in function, and its rise can lag actual kidney injury by several days. A more accurate means of rapid and accurate detecting changes in renal function early in the course of HRS that associate with outcomes may allow for more prompt initiation of therapy and improved outcomes.

Cystatin C (CysC) is a low-molecular-weight cysteine proteinase inhibitor synthesized at a constant rate by all nucleated cells. CysC is freely filtered by the glomerulus, almost completely reabsorbed and catabolized by the proximal tubule, and does not undergo secretion. CysC levels are less influenced by nonrenal factors than Scr and it has thus been proposed as a superior marker of glomerular filtration. In HRS, CysC rises more rapidly than Scr in some settings and has been shown to associate more strongly with outcomes. CysC performs better than Scr in early detection of HRS. CysC is associated with duration of HRS, need for renal replacement therapy, and short and long term mortality in HRS.

The research was aimed to investigate the use of cystatin C (CysC) for early detection of HRS in cirrhotics. CysC, a low-molecular-weight cysteine proteinase inhibitor, is a potentially more accurate marker of glomerular filtration. We conducted a prospective multicenter study in patients with ALC, comparing changes in CysC and Scr immediately following onset of HRS as predictors of a composite endpoint of dialysis or mortality. The results of our study confirmed, that CysC has demonstrated less variability between samples than Scr. Patients were stratified into four groups reflecting changes in Scr and cystatin: both unchanged or decreased 38 (36%) (Scr-/CysC-); only cystatin increased 25 (24%) (Scr-/CysC+); only Scr increased 15 (14%) (Scr+/CysC-); and both increased 28 (26%) (Scr+/CysC+). With Scr-/CysC- as the reference, in both instances where cystatin rose, Scr-/CysC+ and Scr+/CysC+, the primary outcome was significantly more frequent in multivariate analysis, and, respectively. However, when only Scr rose, outcomes were similar to the reference group. Summarizing all above, we can conclude, that changes in CysC levels early in HRS are more closely associated with eventual dialysis or mortality, than Scr and may allow more rapid identification of patients at risk for adverse outcomes.

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#### **PATOGENETIC SIGNIFICANCE OF ENTEROPATHETIC ESCHERICHIA COLI SEROVARIENTS IN THE ENTROCOLITIS CLINICAL FEATURES**

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The aim of the study was to investigate the level and role of enteropathogenic intestinal *Escherichia coli* (*E. coli*) in the development of acute enterocolitis.

The taxonomic composition and microbiota population level in colon cavity content of the 95 patients with diarrheal escherichiosis and 87 healthy individuals were investigated. The age of the patients varied from 25 to 52 years (on the average  $38,66 \pm 3,11$  years). There were 62 women (65,26%) and 33 men (34,74%) among the examined. Colonies of microorganisms have been studied visually in the batch of fresh faeces (not more than 2 hours) of the colon cavity content by a microbiological method.

It was found that the acute diarrheal escherichiosis develops on the background of taxonomic composition and bacterial population violations of autochthonous obligate anaerobic gram-positive microbiota (*Bifidobacterium* and *Lactobacillus*), with a growing number of bacterial genera *Bacteroides*, *Peptostreptococcus*, *Clostridium* and facultative anaerobic and aerobic bacteria of the genus *Escherichia*, *Proteus*, *Staphylococcus* and fungi genus *Candida*, contamination and colonization of colon cavity conditionally pathogenic enterobacteria (*Citrobacter*, *Enterobacter*, *Proteus*, etc.) *Peptococcus*, fungi genus *Candida*, and the growth and proliferation of Enteropathogenic, Enteroinvasive, Enterohemorrhagic *Escherichia coli* and Hemolytic (*E. coli* Hly +) and lactose-negative *E. coli*.

Dysbacteriosis was diagnosed in 57,89% patients with acute enterocolitis, dysbiosis was diagnosed in 42,11%. Among them, 63,16% subjects had dysbiosis 3<sup>rd</sup> and 4<sup>th</sup> degrees, 36,84% – dysbiosis 1<sup>st</sup> and 2<sup>nd</sup> degrees ( $\chi^2 = 13,16$ ;  $p < 0,001$ ). Among healthy individuals persons with normal flora dominate over those with 1<sup>st</sup> and 2<sup>nd</sup> degrees dysbiosis (89,66% vs. 10,34%,  $p < 0,001$ ).

Pathogenic agents of the acute diarrheal escherichiosis are pathogenic enterobacteria in 41,05% of patients, Enteropathogenic *Escherichia coli* in 29,45% of patients; Enteroinvasive *Escherichia coli* in 23,16% of patients and Enterohemorrhagic *Escherichia coli* in 11,58% of patients.

Clinical manifest of the acute diarrheal escherichiosis is polymorphic and depends on both the biology of the pathogenic agent and on the immunologic status of the macroorganism: in 90 (94,79%) of patients the disease was caused by one serovariant of *E.coli* whereas in 5 (5,26%) – the disease was due to association of enteropathogenic *Escherichia coli* of 2 different taxons. The acute colienteritis was diagnosed in 57 (60,0%) patients, dysenteric type of the disease was diagnosed in 24 (25,26%), choleric type of colienteritis was diagnosed in 14 (14,74%) patients.