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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

GEORGIAN MEDICAL NEWS

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GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board since 1994. GMN carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

GMN is indexed in MEDLINE, SCOPUS, PubMed and VINITI Russian Academy of Sciences. The full text content is available through EBSCO databases.

GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения. Журнал индексируется в MEDLINE, отражён в базе данных SCOPUS, PubMed и ВИНИТИ РАН. Полнотекстовые статьи журнала доступны через БД EBSCO.

GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

ჟურნალი ინდექსირებულია MEDLINE-ის საერთაშორისო სისტემაში, ასახულია SCOPUS-ის, PubMed-ის და ВИНИТИ РАН-ის მონაცემთა ბაზებში. სტატიების სრული ტექსტი ხელმისაწვდომია EBSCO-ს მონაცემთა ბაზებიდან.

WEBSITE

www.geomednews.com

К СВЕДЕНИЮ АВТОРОВ!

При направлении статьи в редакцию необходимо соблюдать следующие правила:

1. Статья должна быть представлена в двух экземплярах, на русском или английском языках, напечатанная через **полтора интервала на одной стороне стандартного листа с шириной левого поля в три сантиметра**. Используемый компьютерный шрифт для текста на русском и английском языках - **Times New Roman** (**Кириллица**), для текста на грузинском языке следует использовать **AcadNusx.** Размер шрифта - **12**. К рукописи, напечатанной на компьютере, должен быть приложен CD со статьей.

2. Размер статьи должен быть не менее десяти и не более двадцати страниц машинописи, включая указатель литературы и резюме на английском, русском и грузинском языках.

3. В статье должны быть освещены актуальность данного материала, методы и результаты исследования и их обсуждение.

При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

5. Таблицы необходимо представлять в печатной форме. Фотокопии не принимаются. **Все цифровые, итоговые и процентные данные в таблицах должны соответствовать таковым в тексте статьи**. Таблицы и графики должны быть озаглавлены.

6. Фотографии должны быть контрастными, фотокопии с рентгенограмм - в позитивном изображении. Рисунки, чертежи и диаграммы следует озаглавить, пронумеровать и вставить в соответствующее место текста **в tiff формате**.

В подписях к микрофотографиям следует указывать степень увеличения через окуляр или объектив и метод окраски или импрегнации срезов.

7. Фамилии отечественных авторов приводятся в оригинальной транскрипции.

8. При оформлении и направлении статей в журнал МНГ просим авторов соблюдать правила, изложенные в «Единых требованиях к рукописям, представляемым в биомедицинские журналы», принятых Международным комитетом редакторов медицинских журналов - http://www.spinesurgery.ru/files/publish.pdf и http://www.nlm.nih.gov/bsd/uniform_requirements.html
В конце каждой оригинальной статьи приводится библиографический список. В список литературы включаются все материалы, на которые имеются ссылки в тексте. Список составляется в алфавитном порядке и нумеруется. Литературный источник приводится на языке оригинала. В списке литературы сначала приводятся работы, написанные знаками грузинского алфавита, затем кириллицей и латиницей. Ссылки на цитируемые работы в тексте статьи даются в квадратных скобках в виде номера, соответствующего номеру данной работы в списке литературы. Большинство цитированных источников должны быть за последние 5-7 лет.

9. Для получения права на публикацию статья должна иметь от руководителя работы или учреждения визу и сопроводительное отношение, написанные или напечатанные на бланке и заверенные подписью и печатью.

10. В конце статьи должны быть подписи всех авторов, полностью приведены их фамилии, имена и отчества, указаны служебный и домашний номера телефонов и адреса или иные координаты. Количество авторов (соавторов) не должно превышать пяти человек.

11. Редакция оставляет за собой право сокращать и исправлять статьи. Корректура авторам не высылается, вся работа и сверка проводится по авторскому оригиналу.

12. Недопустимо направление в редакцию работ, представленных к печати в иных издательствах или опубликованных в других изданиях.

При нарушении указанных правил статьи не рассматриваются.

REQUIREMENTS

Please note, materials submitted to the Editorial Office Staff are supposed to meet the following requirements:

1. Articles must be provided with a double copy, in English or Russian languages and typed or compu-ter-printed on a single side of standard typing paper, with the left margin of **3** centimeters width, and **1.5** spacing between the lines, typeface - **Times New Roman** (**Cyrillic**), print size - **12** (referring to Georgian and Russian materials). With computer-printed texts please enclose a CD carrying the same file titled with Latin symbols.

2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

8. Please follow guidance offered to authors by The International Committee of Medical Journal Editors guidance in its Uniform Requirements for Manuscripts Submitted to Biomedical Journals publication available online at: http://www.nlm.nih.gov/bsd/uniform_requirements.html
http://www.icmje.org/urm_full.pdf
In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

9. To obtain the rights of publication articles must be accompanied by a visa from the project instructor or the establishment, where the work has been performed, and a reference letter, both written or typed on a special signed form, certified by a stamp or a seal.

10. Articles must be signed by all of the authors at the end, and they must be provided with a list of full names, office and home phone numbers and addresses or other non-office locations where the authors could be reached. The number of the authors (co-authors) must not exceed the limit of 5 people.

11. Editorial Staff reserves the rights to cut down in size and correct the articles. Proof-sheets are not sent out to the authors. The entire editorial and collation work is performed according to the author's original text.

12. Sending in the works that have already been assigned to the press by other Editorial Staffs or have been printed by other publishers is not permissible.

Articles that Fail to Meet the Aforementioned Requirements are not Assigned to be Reviewed.

ავტორთა საყურადღებოდ!

რედაქციაში სტატიის წარმოდგენისას საჭიროა დავიცვათ შემდეგი წესები:

1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

3. სტატიაში საჭიროა გაშუქდეს: საკითხის აქტუალობა; კვლევის მიზანი; საკვლევი მასალა და გამოყენებული მეთოდები; მიღებული შედეგები და მათი განსჯა. ექსპერიმენტული ხასიათის სტატიების წარმოდგენისას ავტორებმა უნდა მიუთითონ საექსპერიმენტო ცხოველების სახეობა და რაოდენობა; გაუტკივარებისა და დაძინების მეთოდები (მწვავე ცდების პირობებში).

4. სტატიას თან უნდა ახლდეს რეზიუმე ინგლისურ, რუსულ და ქართულ ენებზე არანაკლებ ნახევარი გვერდის მოცულობისა (სათაურის, ავტორების, დაწესებულების მითითებით და უნდა შეიცავდეს შემდეგ განყოფილებებს: მიზანი, მასალა და მეთოდები, შედეგები და დასკვნები; ტექსტუალური ნაწილი არ უნდა იყოს 15 სტრიქონზე ნაკლები) და საკვანძო სიტყვების ჩამონათვალი (key words).

5. ცხრილები საჭიროა წარმოადგინოთ ნაბეჭდი სახით. ყველა ციფრული, შემაჯამებელი და პროცენტული მონაცემები უნდა შეესაბამებოდეს ტექსტში მოყვანილს.

6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

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SPECIFICITIES OF THE COURSE OF SUBCLINICAL HEPATITIS AMONG YOUNG ADULTS WITH ACUIE GLOMERULONEPHRITIS

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Abstract.

In spite of the progress in medical science in our country during recent years, the investigation of some problems of development and course of acute glomerulonephritis (AG) particularly in young adults remains topical. In this paper we discuss classical types of AG in young adults, when the intake of paracetamol and diclofenac led to a dysfunctional and organic liver injury, at the same time it negatively affected the course of AG.

Goal: Assessment of cause-and-effect relations of renal and liver injuries in young adults with acute glomerulonephritis.

Methods: To achieve the goals of the research we examined 150 male patients with AG, aged 18-25. According to clinical presentations all the patients were divided into two groups. In the first group (102 patients) the disease manifested with acute nephritic syndrome; in the second group of patients (48 patients) - with isolated urinary syndrome.

Results: Out of 150 patients examined for AG 66 had subclinical liver injury, which resulted from the effect of antipyretic hepatotoxic drugs taken in the initial stage of the disease. Due to the toxic and immunological liver injury, levels of transaminases increase, and albumin levels decrease. These changes occur along with the development of AG and are correlated with some laboratory values (ASLO, CRP, ESR, hematuria), the injury is more evident when etiological factor is the streptococcal infection.

Conclusion: In AG liver injury has a toxic allergic character and is more expressed in post streptococcal glomerulonephritis. Frequency of liver injury depends on specific features of a particular organism; it does not depend on the dose of the taken drug. In case of any type of AG it is necessary to assess the functional state of liver and after the treatment of the main disease hepatologist follow-up of patients is recommended.

Key words. Acute glomerulonephritis, acute nephritic syndrome, isolated urinary syndrome, liver injury, hypertransaminasemia.

Introduction.

Acute diffuse glomerulonephritis is an immuno-inflammatory kidney disease, which manifests mainly with the primary lesion of kidney glomeruli and involvement of all renal structural elements in the pathological process, which is clinically manifested by renal and extrarenal symptoms [1-3]. In spite of the progress in medical science in our country in recent years, the investigation of some problems of development and course of acute glomerulonephritis particularly in young adults remains actual.

A number of investigations show that acute glomerulonephritis occurs mainly in male patients aged 15-19, this age group also corresponds to the military service call-up age [4,5]. In young men adaptation stress, change of everyday life conditions and food, long hours of mandatory parade drilling, heavy physical load lead to the suppression of the body immune system, increase the likelihood of streptostaphylococcal infection, which can lead to the development of acute glomerulonephritis.

The main etiological factor of the disease is the streptococcal infection in the patient (tonsilitis, pharyngitis, exacerbation of chronic tonsilitis, scarlet fever, erysipelas, etc..). Acute glomerulonephritis is frequently caused by 12 and 49 species of β-hemolytic streptococcus of group A (poststreptococcal glomerulonephritis) [6,7]. Other possible etiological factors are Hepatitis B virus (causes mainly membranous nephritis), measles, infectious mononucleosis and herpes viruses, sometimes acute glomerulonephritis can develop after staphylococcal and pneumococcal infections [8,9]. The disease develops within 7-20 days of the influence of above-mentioned factors, as a rule it develops with a rise of body temperature, for which patients take nonsteroidal anti-inflammatory drugs, paracetamol, which have nephrotoxic and hepatotoxic effect. Liver plays a major role in the metabolism of various drugs especially when taken orally. Drug lesions of liver occur more frequently than they are diagnosed. Two types of liver lesions are differentiated – toxic and idiosyncratic, which in its turn can be immuno-allergic and metabolic. The idiosyncratic type of liver lesion is characterized by the isolated increase serum transaminases (AST, ALT). Toxic lesion of liver is manifested by the decrease of albumin synthesis, which later leads to the decrease of drug binding property of liver, including antipyretic drugs. The biological activity of the drug and the biotransformation of its toxic metabolites increase [5,10].

In the literature quantity dependent and quantity nondependent types of liver drug lesions are described, the latter is more unpredictable and occurs rarely. That type of liver lesion develops as a slow hypersensitivity reaction. Liver drug lesion develops within 5-25 days after the drug intake [2,9].

In the literature sources available to us there are no data concerning primary liver lesion in renal pathologies. In the current paper we discuss classical types of acute glomerulonephritis, when the intake of paracetamol and diclofenac resulted in a dysfunctional and organic liver lesion, at the same time it negatively affected the course of acute glomerulonephritis.

Goal: Assessment of cause-and-effect relations of renal and liver lesions in young adults with acute glomerulonephritis.

Objectives: The study was conducted among patients hospitalized for acute glomerulonephritis at the RA Central Clinical Military Hospital. The patients gave their verbal consent to participate in the study, no ethical committee opinion was needed, given the comprehensive analysis of protocol-based examination results.

Assessment of the degree of renal and liver lesions related to the character of development factors of acute glomerulonephritis. Assessment of hepatotoxic effect of the medication used in the early stages of the disease. Assessment of correlations between

clinical signs and laboratory values and degree of liver lesion in acute glomerulonephritis.

Methods.

To achieve the goals of the research 150 male patients aged 18-25 were examined, who were hospitalized and treated in Central Clinical Military Hospital of Yerevan. All the patients were examined according to the accepted examination schemes: complete blood count and urinalysis, 24-hour urinalysis, biochemical blood examination – glucose, total protein, albumin, bilirubin, aspartate aminotransferase (AST), alanine aminotransferase (ALT), C-reactive protein, antistreptolysin O, creatinine, urea, lipid profile, coagulation tests, C3 and C4 fractions of complement, glomerular filtration rate, abdominal ultrasound, chest X-ray, ECG, echocardiography. Acute glomerulonephritis was diagnosed based on history, clinical and instrumental investigations. In history a special significance is assigned to the risk factors and etiological factor. In risk factors, the presence of cold and vaccination are specified. The history of all 150 patients includes streptococcal infection (84 patients) or viral factor (66 patients), which was manifested by increase in temperature. To lower the body temperature, the patients were given nonsteroidal anti-inflammatory drugs, paracetamol, and diclofenac in particular. Average intake period of drugs lasted 3-5 days. All the patients were hospitalized within 7-20 days of the disease with the diagnosis of acute glomerulonephritis. In course of the medical examination, the mean of individual indicators and its probable error were determined. The reliability of the results was assessed by Student's t-distribution.

Results.

The description of clinical manifestations and instrumental examinations of the patients is presented in Table 1.

As the table shows the patients mainly complained of low back dull pain, headache, dyspnea, decrease of 24-hour urine output, urine colour darkening, general weakness. According to the objective examination 67.9% of patients had pastose faces or face swelling, 67.6% - lower limb edema of various presentation, 83.3% developed an increase of arterial pressure. 68% had ascites, 65.3%-hydrothorax, 34.5– hydropericarditis. According to clinical presentations all patients were divided into two groups: in the first group (102 patients) the disease manifested with acute nephritic syndrome – swelling, hypertonia, urinary syndrome. In the second group of patients (48 patients) the disease manifested with isolated urinary syndrome. In 66 patients signs of liver lesion were detected. In 48 patients the disease was complicated by acute renal failure.

Further study involved 66 patients with signs of liver lesion (in this group of patients, the ultrasound examination revealed an increase in the size of the liver, lab tests indicated a double or more increase in the amount of AST and ALT). According to the clinical forms in 44 of these patients, the disease developed with acute nephritic syndrome, in 22 - isolated urinary syndrome with hematuria. The current conditions of oligoanuria, hypercreatinemia, and high levels of BUD served as a basis for diagnosing acute renal failure.

The results of laboratory examination of acute glomerulonephritis with acute nephritic syndrome and liver lesion and hematuric form are presented in Table 2.

Table 1. *The picture of complaints, objective examination and some instrumental investigations of patients with acute glomerulonephritis.*

Data		Patients group 150	
		n	% - according to the number of patients
Low back dull pain		130	86.6
Headache		88	58.6
Dyspnea		76	50.6
Decrease of 24-hour urine output		110	73.3
Urine colour darkening		146	97.3
Chest pain		20	13.3
General weakness		78	52
Hemoptysis		12	8
Pale skin		121	80.6
Enlarged peripheral lymph nodes		8	5.3
Pastose face		50	33.3
Face edema		52	34.6
Lower extremity oedema	Mild	28	18.6
	Moderate	42	28
	Considerable	32	21.3
Pulse	Bradycardia	69	49
	Tachycardia	25	16.6
	Normal rhythm	56	37.3
Arterial blood pressure	140-150/90-100	62	41.3
	160-170/100-110	48	32
	>170/100	15	10
Heart sounds	muffled	38	25.3
	Apex murmur	91	60.6
	Normal	21	14
Auscultation of lungs	Vesicular breathing	33	22
	Wet rales of various calibre	29	19.3
	Diminished vesicular sounds in lower lung fields	88	58.6
ECG	Mitral insufficiency	102	68
	Tricuspid insufficiency	93	62
	Pulmonary hypertension	33	22
	Hydropericarditis	52	34.6
Ultrasound	Hepatomegaly	53	35.3
	Splenomegaly	13	8.6
	Diffuse renal changes	145	0.96
	Ascites	102	68
Chest X-ray	Normal	31	20.6
	Pneumonia	21	14
	Hydrothorax	98	65.3

Proteinuria data in urinalysis and 24-hour urine in acute glomerulonephritis are presented in Table 3.

Peripheral blood analysis shows moderate anemia in 22 patients with isolated urinary syndrome and leukocytosis in some patients with acute nephritic syndrome. Increase in erythrocyte sedimentation rate (ESR) is present almost in all patients and is more evident in patients with acute nephritic syndrome. The concentration function of kidneys was preserved in all the patients. Proteinuria in 24-hour urine fluctuated within

Table 2. *The results of laboratory examination of acute glomerulonephritis with acute nephritic syndrome and liver lesion and hematuric form.*

Laboratory values	Normal	Acute nephritic syndrome 44 patients		Hematuric form 22 patients	
		Value at hospitalization M±m	Value at discharge from the hospital M±m	Value at hospitalization M±m	Value at discharge from the hospital M±m
Hemoglobulin	13.1-16.7g/dl	13.0±1.85 p<0,05	14.0±4.37 p<0,25	11.5±0.98 p<0,25	12.02±2.89 p<0,05
Erythrocyte	4.24-5.72×10/ml	4,56±0,03 p<0,01	4,6±0,14 p<0,05	3,99±0.15 p<0,01	4,18±0,11 p<0,05
Leucocyte	3.8-11.2×10/ml	10,98±2,25 p<0,01	7,66±2,15 p<0,05	8,18±0,24 p<0,01	7,44±0,23 p<0,05
ESR	2.0-10.0mm/h	38,8±1.55 p<0,01	14,16±1,01 p<0,02	28,01±1,81 p<0,01	12,8±1.89 p<0,02
CRP	<5mg/l	28,26±0.12 p<0,25	5,3±4.37 p<0,25	26,16±0.25 p<0,25	6,1±2.81 p<0,25
ASLO	<200unit/l	640±50.85 p<0,01	240±4.37 p<0,01	400±20.98 p<0,01	228.02±20,1 p<0,01
Complement C3 fraction	0.84-1.67g/l	0,27±0,15 p<0,05	0,83±0,26 p<0,05	0,44±0.16 p<0,05	0,79±0,08 p<0,05
Complement C4 fraction	0.1-0.4g/l	0,17±0,02 p<0,05	0,21±0,01 p<0,05	0,23±0.01 p<0,05	0,21±0,02 p<0,05
AST	<37/31unit/l	88,9±5.85 p<0,05	39,3±3.37 p<0,05	78,12±5.48 p<0,05	48,3±3.29 p<0,05
ALT	<42/32unit/l	120,1±5.85 p<0,05	44±6.37 p<0,05	95,4±5.98 p<0,02	42,3±6.29 p<0,02
Albumin	34-48g/l	18,18±4.65 p<0,07	33,12±1.77 p<0,08	26,49±2,51 p<0,07	36,12±1.89 p<0,08
Creatinine	62-106mcmol/l	125,1±28.8 p<0,25	88±12.86 p<0,25	115,7±25.9 p<0,025	62,3±13.2 p<0,025
Fibrinogen	1.8-3.5g/l	4,8±1.85 p<0,05	3,26±3,9 p<0,05	4,3±2,6 p<0,05	2,2±1.29 p<0,05

Table 3. *Proteinuria data in acute glomerulonephritis.*

Complete urinalysis	Normal	Acute Nephritic syndrome 44 patients		Hematuric form 22 patients	
		Value at hospitalization M±m	Value at discharge from the hospital M±m	Value at hospitalization M±m	Value at discharge from the hospital M±m
Proteinuria in 24-hour urine	<0,033‰	2,82±0,24 p<0,01	0.033±0,001 p<0,01	1,11±0,64 p<0,01	0.033±0,018 p<0,01
Proteinuria	<0,10g/l	0,86±0,2 p<0,01	0,36±0,08 p<0,01	0,81±0,11 p<0,01	0,18±0,02 p<0,01

Table 4. *Analysis of values of liver lesions in acute glomerulonephritis caused by various etiological factors.*

Etiological factor	Number of patients	ALT	AST	Albumin
Streptococcal infection	42	121.1±11.85	98.18±5.6	22.01±2.26
Viral infection	24	91.3±11.8	76.2±6.3	29.6±3.85
Total	66	p<0,05	p<0,25	p<0,05

the limits of 0.86-2.82g/l. At discharge from the hospital, the patients mainly had slightly manifested trace proteinuria. At hospitalization 36 patients had macrohematuria (24 patients with acute nephritic syndrome, 12 patients with isolated urinary syndrome). The other 10 patients had microhematuria, with various extent of presentation. At discharge from the hospital 12 patients out of 66 still had mild microhematuria. 21 patients out of 66 developed acute renal failure. 16 patients out of 21 were hospitalized in oliguric stage, 5 – in the stage of diuresis recovery. In the other 45 patients' nitrogen excreting and filtration functions of kidneys were preserved. In 66 patients' clinical signs of liver lesion were missing. Ultrasound examination detected slight liver enlargement (2-3cm), with moderate changes in parenchyma. Laboratory examinations showed increase in AST and ALT values and decrease in albumin. 44 patients had increased AST and ALT values: 120.1±5.85 unit/l

and 88.9±5.85 unit/l respectively accompanied by the decrease of blood albumin – 18.18±4.65 g/l. In 22 patients with isolated urinary syndrome, the increase of transaminases and the decrease of albumin level were comparatively less expressed than in cases with acute nephritic syndrome, and were as follows: 95.4±5.98, 78.12±5.48, 26.49±2.51. In all the patient's laboratory changes had subclinical course.

In patients with liver lesion the comparison of other laboratory data (complement C fraction, antistreptolysin O, C-reactive protein values) with the clinical presentation of the disease shows that the decrease of complement C fraction and the increase of antistreptolysin O and C-reactive protein are more expressed in acute glomerulonephritis with acute nephritic syndrome (Table 2).

During the study the correlation between liver functional activity indicators with the values of antistreptolysin O, C-reactive protein, erythrocyte sedimentation rate was assessed. The study shows that there is direct correlation between AST, ALT, and albumin with antistreptolysin O (ASLO), C-reactive protein and erythrocyte sedimentation rate (ESR) values, which are more expressed in acute nephritic syndrome. In the next stage of the study a comparative analysis of values of liver lesions in acute glomerulonephritis caused by various etiological factors was carried out. The results are presented in Table 4.

As the table shows liver lesions are mainly manifested in acute glomerulonephritis caused by streptococcal infection.

Discussion.

Thus, out of 150 patients examined for acute glomerulonephritis 66 had subclinical liver lesion, resulted from the effect of antipyretic hepatotoxic drugs taken in the initial stage of the disease. Hepatocyte injury results either from the immediate effect of metabolic products drugs or infection factors or from their indirect effect [11-13]. In the body they are considered as neoantigens, as a result antibodies are synthesized towards them and the created antigen-antibody complex also damages hepatocytes, as well as endothelial and mesangial cells in kidneys. In fact, it is not excluded that common mechanisms of acute glomerulonephritis and liver injury exist [14,15]. Our studies showed that the frequency of liver lesion depends on specific features of a particular organism, which cannot be predicted. 139 of the 150 patients involved in this study received non-steroidal anti-inflammatory drugs for an average of 3 – 5 days to beat the fever, however, only 66 revealed subclinical liver damage. Based on the above, we can conclude that the frequency of liver damage depends on the individual characteristics of an individual organism, which cannot be predicted. The manifestation of liver lesion is not related to the dose of the taken drug. Toxic and immunologic lesion of liver leads to the subclinical increase of transaminases and decrease of albumin levels. These changes happen along with the development of acute glomerulonephritis and are correlated with some laboratory values (ASLO, CRP, ESR, hematuria), the lesion is more evident when etiological factor is the streptococcal infection.

Conclusion.

1. In acute glomerulonephritis liver lesions have subclinical course and manifest with the increase of level of transaminases and decrease of albumin level.

2. There is correlation between clinical presentation of acute glomerulonephritis and laboratory values and extent of liver injury. There is direct correlation between the increase of transaminases and extent of hematuria, ASLO, CRP, ESR values.

3.There is no evident correlation between the manifestation of hypoalbuminemia and proteinuria. The manifestation of hypoalbuminemia is connected with the extent of liver injury.

4. There is direct correlation between the extent of liver lesion and etiological factor of acute glomerulonephritis. In streptococcal infection the indicators of liver lesion are more expressed.

5. The reaction of liver in the form of hypoalbuminemia and hypertransaminasemia towards antipyretic drugs does not depend on the drug dose.

6. In acute glomerulonephritis liver lesion has a toxicoallergic character, it develops as a result of the effect of antipyretic drug metabolites, infection factor and immunologic complexes.

Practical Recommendations.

In case of viral and bacterial infections antipyretic drugs should be administered along with hepatoprotectors. In case of any type of acute glomerulonephritis it is necessary to assess the functional state of liver. In case of acute glomerulonephritis with liver lesion it is recommended to perform the assessment of the functional state of liver and dynamic control during a 5-year period after recovery.

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Резюме

Особенности течения субклинического гепатита у лиц молодого возраста с острым гломерулонефритом

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Резюме: Несмотря на прогресс в медицинской науке в нашей стране, исследование проблем развития и течения острого гломерулонефрита (ОГ), в особенности у лиц молодого возраста, остается актуальным. В данной работе нами обсуждаются классические формы ОГ у лиц молодого возраста, при которых применение парацетамола и диклофенака привело к дисфункциональному и органическому поражению печени, а также имело отрицательное воздействие на динамику ОГ. **Цель:** Оценить причинно-следственные связи поражений почек и печени у лиц молодого возраста при ОГ. **Методы:** Обследовано 150 больных мужского пола в возрасте 18-25 лет, которые были госпитализированы с диагнозом ОГ. Больные были разделены на две группы исходя из клинических проявлений болезни. У больных первой группы (102 больных) болезнь проявилась острым нефритическим синдромом, у больных второй группы (48 больных)- изолированным мочевым синдромом. **Результаты:** Из 150 больных у 66 было обнаружено субклиническое поражение печени, обусловленное гепатотоксическим воздействием жаропонижающих препаратов, принятых на начальной стадии заболевания. Токсическое и иммунное поражение печени приводит к повышению уровня трансаминаз, снижению уровня альбумина. Эти изменения протекают параллельно с развитием ОГ и коррелируют с некоторыми лабораторными показателями (АСЛО, СРБ, СОЭ, гематурия), поражение более выражено, когда этиологическим фактором является стрептококковая инфекция. Заключение: При ОГ поражение печени носит токсико-аллергический характер, оно более выражено при постстрептококковых гломерулонефритах. Частота поражения печени зависит от особенностей конкретного организма, она не зависит от дозы принятого препарата. При любых формах ОГ необходимо оценить функциональное состояние печени, после излечения основного заболевания рекомендуется находиться под наблюдением гепатолога.

Ключевые слова: острый гломерулонефрит, острый нефритический синдром, изолированный мочевой синдром, поражение печени, гипертрансаминаземия.