STUDY OF LABORATORY AND CLINICAL EXAMINATIONS OF GASTROINTESTINAL TRACT DAMAGE IN SYSTEMIC LUPUS RED

Yuldasheva Sayyora Khudayberganovna
Artikov Hikmat Kahramon ugli
Department of "internal diseases, rehabilitation and folk medicine" Urgench branch of the Tashkent Medical Academy, Republic of Uzbekistan. Urgench

TIZIMLI QIZIL YUGURUKDA MEDA-ICHAK TRAKTINING ZARARLANISHINI LABORATORIYA VA KLINIK TAHLILLARINI O'RGANISH

Yuldasheva Sayyora Xudayberganovna
Artikov Hikmat Kahramon o'g'li
Toshkent tibbiyot akademiyasi Urganch filiali "Ichki kasalliklar, reabilitologiya va xalq tabobati" kafedrasi, O'zbekiston Respublikasi. Urganch

ИЗУЧЕНИЕ ЛАБОРАТОРНО-КЛИНИЧЕСКИХ ИССЛЕДОВАНИЙ ПОРАЖЕНИЯ ЖЕЛУДОЧНО-КИШЕЧНОГО ТРАКТА ПРИ СИСТЕМНОЙ КРАСНОЙ ВОЛЧАНКЕ

Юлдашева Сайёра Худайберганoвна
Артиков Хикмат Кахрамон угли
Кафедра «Внутренних болезней, реабилитологии и народной медицины» Ургенчского филиала Ташкентской медицинской академии, Республика Узбекистан. Город Ургенч.

Аннотация. В этой статье рассматриваются анализы и клинические поражения желудочно-кишечного тракта при системной красной волчанке. В последние годы увеличилась ранняя диагностика, в результате чего болезнь диагностируется у 500-600 человек. Системная красная волчанка - сложное заболевание без известной причины. Скорее всего, это не одна причина, а комбинация нескольких факторов, таких как генетика, окружающая среда и возможно, гормональные факторы, сочетание которых может привести к заболеванию. В настоящее время системная красная волчанка является неизлечимым заболеванием.

Ключевые слова: генетические факторы, гормональные изменения, аутоиммунные заболевания, антитела, аутоантитела, кортикостероидные гормоны.

Annotation: In this article, analyzes and clinical lesions of the gastric-intestinal tract in systemic lupus erythematosus were studied. In recent years, there has been an increase in early diagnosis, as a result, the disease is diagnosed in 500-600 patients per 1 million population. Probably, this apparent increase is due to improved diagnostic capabilities, primarily due to the development of new immunological criteria. Lupus is a topic of much research as scientists try to determine what causes lupus and how best to treat it.

Key words: genetic factors, hormonal changes, autoimmune disease, antibodies, autoantibodies, corticosteroid hormones.

Tadqiqotning maqsadi: tizimli qizil yuguruk (TQY) bilan og'rigan bemorlarda asosiy klinik ko'rinishlarni, ayniqsa bemorlarda jigar shikastlanishini o'rganish edi. **Materiallar va usullar:** Biz 17 yoshdan 60 yoshgacha bo'lgan TQY (36 ayol va 14 erkak) bo'lgan 16-60 yoshdagi 50 nafar bemorni kuzatdik. Bemorlarning o'rtacha yoshi 38,5 edi. Bu bemorlarning barchasi dispanser hisobida, Xorazm viloyati ko'p tarmoqli tibbiyot markazining revmatologiya bo'limida muntazam statsionar davolanmoqda.

Natijalar: Laboratoriya diagnostikasi. Tashxis qo'yishda, birinchi navbatda, periferik qonning tarkibiga e'tibor qaratish lozim. TQY, normoxromik anemiya, leykopeniya va ESR keskin o'sishi bilan og'rigan bemorlarning 90% da. Siydik testlarida TQR bilan og'rigan bemorlarning 83% da - turli darajadagi proteinuriya, leykotsitlar, eritrotsitlar - va silindruriya. Siydik oqsillari orasida qo'pol fraktsiyalar ustun bo'lishi xarakterlidir, ya'ni proteinuriya selektiv emas. LE hujayralarining aniqlanishi tipik hisoblanadi. Biroq, hozirgi vaqtda bu hodisa nafaqat qizil yuguruk uchun xarakterli ekanligi va nisbatan kamdan-kam uchraydiganligi ko'rsatilgan. Bemorlarning 48% da LE hujayralari topilgan. Nativ DNKga va antinuklear omilga (ya'ni zardobdagi denaturatsiyalangan DNKga antitelalar) antitellarni aniqlash eng katta ahamiyatga ega. Qon zardobidagi komplementning umumiy gemolitik faolligi uning klassik yo'l bo'ylab faollashishi tufayli kamayadi. TYQ bilan og'rigan bemorlarning 94 foizida mahalliy DNK va antinuklear omilga antikorlar topilgan. Qonning biokimyoviy tahlili: kreatinin, ALAT, ASAT - bemorlarning 25% da, 28% bemorlarda kreatin kinazning ortishi. Plevral efüzyon (plevrit), qizil yuguruk, o'pka emboliyasi bilan og'rigan bemorlarning 38% da ko'krak qafasining rentgen va kompyuter tomografiyasi. Yadro magnit-rezonansi va angiografiya - markaziy asab tizimining 56% lezyonlarida vaskulit aniqlanadi. Exokardiyografiya - bemorlarning 78% perikard bo'shlig'ida suyuqlik, perikard-ning shikastlanishi, yurak klapanlarining shikastlanishi va boshqalarni aniqladi. Ultratovush diagnostikasi bemorlarning 60

foizida jigarning diffuz siqilishini, bemorlarning 35 % da gepatosplenomegaliyani aniqladi.

Xulosa: Tahlillar va klinik tekshiruvlarga ko'ra, terining shikastlanishi kasallikning boshlanishida faqat 20-25% bemorlarda, 60-70% bemorlarda kechroq, 10-15% bemorlarda kasallikning teri ko'rinishida paydo bo'ladi. Teri o'zgarishlari tananing quyoshga ta'sir qiladigan joylarida paydo bo'ladi: yuz, bo'yin, elka. Lezyonlar eritema (peeling bilan qizg'ish plitalar), qirralarning bo'ylab kengaygan kapillyarlar, ortiqcha yoki pigment yetishmasligi bo'lgan joylar ko'rinishiga ega. Yuzda bunday o'zgarishlar kapalakning ko'rinishiga o'xshaydi, chunki burunning orqa qismi va yonoqlari ta'sirlanadi. Soch to'kilishi (alopesiya) kamdan-kam uchraydi, odatda temporal hududlarga ta'sir qiladi. Sochlar cheklangan hududda tushadi. Terining quyosh nuriga sezgirligi (fotosensibilizatsiya) bemorlarning 30-60% da uchraydi. Qizarish, pigmentatsiyaning pasayishi, lablarning noto'g'ri ovqatlanishi (cheilitis), achchiqlanish, depressiya - kamdan-kam hollarda ensefalopatiya (21-67%)

Ovqat hazm qilish traktining klinik lezyonlari SLE bilan og'rigan bemorlarning 20% da tashxis qilinadi. 5% hollarda qizilo'ngachning shikastlanishi, yutish aktining buzilishi, qizilo'ngachning kengayishi kuzatiladi. Me'da va 12-barmoq ichakning yarasi ikkala sababdan kelib chiqadi: kasallikning o'zi va davolashning yon ta'siri, ayniqsa NSAID va kortikosteroidlar bilan davolashdan keyin.

Izoh: Ushbu maqolada tizimli qizil yugurukda oshqozon-ichak traktining tahlillari va klinik shikastlanishlari o'rganildi. So'nggi yillarda erta tashxis qo'yish ko'paydi, natijada kasallik 500-600 bemorga tashxis qo'yilmoqda**.** Tizimli qizil yuguruk - bu noma'lum sababi bo'lmagan murakkab kasallik. Ehtimol, bu bitta sabab emas, balki genetik, atrof-muhit va ehtimol gormonal kabi bir nechta omillarning kombinatsiyasi bo'lib, ularning kombinatsiyasi kasallikka olib kelishi mumkin. Hozirgi vaqtda tizimli qizil yuguruk davolab bo'lmaydigan kasallikdir.

Kalit so'zlar: genetik omillar, gormonal o'zgarishlar, autoimmun kasallik, antikorlar, otoantikorlar, kortikosteroid gormonlar.

Целью исследования явилось изучение основных клинических проявлений у больных с системной красной волчанкой (СКВ), особенно поражения печени у больных.

Материалы и методы: Наблюдали 50 больных с 16-60 лет с СКВ (36 женщин и 14 мужчин) в возрасте от 17 до 60 лет. Средний возраст пациентов составило 38,5. Все эти больные находится в диспансерном учете, регулярно получают стационарное лечения в ревматологическом отделения Хорезмского областного многопрофильного медицинского центра **Результаты.** При постановке диагноза прежде всего надо ориентироваться на состав периферической крови. У 90% больных с СКВ нормохромная анемия,

лейкопения и резкое увеличение СОЭ. У 83% больных с СКВ в анализах мочи - протеинурия разной степени выраженности, лейкоцит, эритроцит и цилиндрурия. Характерно, что среди белков мочи превалируют грубодисперсные фракции, то есть протеинурия не селективна. Типичным считается обнаружение LE-клеток. Однако в настоящее время показано, что этот феномен характерен не только для красной волчанки и встречается к тому же относительно редко. У 48% больных обнаружено LE-клеток. Наибольшее значение имеет определение антител к нативной ДНК и антинуклеарного фактора (то есть антител к денатурированной ДНК в сыворотке крови). Уровень общей гемолитической активности комплемента в сыворотке снижен за счет активации его по классическому пути. У 94 % больных с СКВ обнаружено антитела к нативной ДНК и антинуклеарного фактора. По анализам и клиническим обследованиям поражения кожи в начале заболевания проявляются лишь у 20-25% больных, у 60-70% больных они возникают позже, у 10-15% кожные проявления болезни и вовсе не возникают. Кожные изменения проявляются на открытых для солнца участках тела: лицо, шея, плечи. Поражения имеют вид эритемы (красноватые бляшки с шелушением), по краям расширенные капилляры, участки с избытком или недостатком пигмента. На лице такие изменения напоминают вид бабочки, так как поражается спинка носа и щеки.Выпадение волос (алопеция), проявляется редко, обычно поражая височные области. Волосы выпадают на ограниченном участке. Повышенная чувствительность кожи к солнечным лучам (фотосенсибилизация) возникает у 30-60% больных Покраснение, снижение пигментации, нарушение питания тканей губ (хейлит). Раздражительность, депрессия редко энцефалопатия (21-67%) Клинические поражение пищеварительного тракта диагностируются у 20 % больных СКВ. Поражение пищевода, нарушение акта глотания, расширение пищевода встречается в 5% случаев. Язвы желудка и 12-перстой кишки вызваны как самой болезнью, так и побочными эффектами лечения, особенно после лечения с НПВС и кортикостероидами. Все больные по течениям процесса(острый, подострый, хронический) получают следующие препараты: из цитостатических иммунодепрессантов циклофосфамид (ЦФ) или азатиоприн, метотрексат (по индивидуальной дозе). Больным СКВ с низкой активностью заболевания назначают небольшие дозы ГК (преднизолон < 10 мг/сут), с умеренной активностью – средние дозы (20–40 мг/сут). При наличии тяжелой органной патологии – диффузном поражении центральной нервной системы (ЦНС), волчаночном нефрите, гематологических нарушений (тромбоцитопении, гемолитической анемии), генерализованном поражении кожи – необходимы более высокие дозы ГК – > 40 мг/сут. Особое место в лечении больных СКВ занимают аминохинолиновые производные (АП). Хлорохин фосфата и

гидроксихлорохин сульфата (Плаквенил) в течение многих лет с успехом применялись при СКВ, главным образом при невысокой и умеренной активности болезни. Отличительной особенностью АП является низкая частота побочных реакций, требующих прекращения лечения, при этом токсичность гидроксихлорохина (Плаквенила) в два раза ниже по сравнению с хлорохином. Большие надежды у больных СКВ, безусловно, связаны с ритуксимабом, которые представляет собой рекомбинантные химерные моноклональные антитела к поверхностным рецепторам лимфоцитов – CD20 Блокаторы ФНО-α (Инфликсимаб, Адалимумаб, Этанерцепт). Основная часть больных получали монотерапию рпитуксимабом (4 инфузии в неделю из расчета 375 мг/м2), и 30% – сочетание препарата с ЦФ. В целом лечение ритуксимабом приводило к достоверному снижению активности заболевания более чем у 80%. Клинический эффект ритуксимаба сопровождался положительной динамикой морфологических изменений в клубочках по данным повторных биопсий.

Relevante: Systemic lupus erythematosus is a complex disease with no known cause. It is likely that this is not a single cause, but rather a combination of several factors, including genetic, environmental and possibly hormonal factors, the combination of which can cause the disease. Currently, systemic lupus erythematosus is an incurable disease [4]. This disease is now considered a model of autoimmune disease. Therefore, understanding the many mechanisms of disease in SLE is key to understanding the immune disorders that occur in many human diseases [8]. Some of the questions scientists are working on include: what exactly causes lupus and why? Why do women get sick more often than men? Why are there more cases of lupus in some racial and ethnic groups? What is disturbed in the immune system and why? How can we correct the functions of the immune system when it is disturbed? How to treat to reduce or cure the symptoms of lupus? Currently, SLE is an incurable disease [10]. Due to the constant use of drugs, a long-term remission is achieved. SLE occurs predominantly in young (20-30 years old) women, but cases of the disease are not uncommon in adolescents and older people (over 40-50 years old) [1]. Among the sick, only 10% of men are noted, but the disease is more severe in them than in women. Provoking factors are often insolation, drug intolerance, stress; in women - childbirth or abortion. Liver dysfunction is not considered the main organ pathology in SLE [9], and liver dysfunction is not included in the classification and diagnostic criteria for SLE. The incidence of liver dysfunction or abnormal liver enzyme values during SLE ranged from 19% to 60% [10]. Moreover, the prevalence of hepatomegaly in patients with SLE ranged from 12% to 55%. Fluctuations in the values of alanine transaminase, corresponding to the activity of SLE, were noted in some patients with SLE [7]; however, no correlation between SLE activity and incidence of liver disease was found in any of the cases reviewed [6]. This discrepancy may be due to different causes of

liver dysfunction or abnormal liver enzyme values in patients with SLE. To date, there have been few systematic reviews of the literature on concomitant cases of SLE and PKD, and the clinical features and pathophysiology of associated cases of SLE and PKD remain unclear. However, it may be necessary to distinguish between complications of PBC in patients with SLE involving the liver because some patients with PBC may progress to occult end-stage liver failure, and long-term use of glucocorticosteroids for SLE is potentially associated with significant side effects, including osteoporosis, which varies. from 20% to 44% in patients with PBS, increasing with the progression of PBP [8]. In contrast, the liver is the target of autoimmune reactions, as seen in autoimmune diseases such as autoimmune hepatitis (AIH), primary biliary cirrhosis (PBC), and primary sclerosing cholangitis (PSC). Liver involvement in SLE patients includes several liver diseases or SLE itself, and it is necessary to distinguish between causes of liver dysfunction or abnormal liver enzyme values. However, it is sometimes difficult to distinguish between causes of liver injury in patients with SLE, and the use of immunosuppressive therapy for SLE may confound clinical and laboratory findings about the underlying autoimmune liver disease, leading to a diagnostic delay or occult progression of liver cirrhosis (LC) [2].

Purpose: The aim of the study was to study the main clinical manifestations in patients with systemic lupus erythematosus (SLE), especially liver damage in patients.

Materials and Methods: We observed 50 patients aged 16-60 years with SLE (36 women and 14 men) aged 17 to 60 years. The average age of the patients was 38.5. All these patients are registered in the dispensary, regularly receive inpatient treatment in the rheumatology department of the Khorezm regional multidisciplinary medical center.

Laboratory diagnostics. When making a diagnosis, first of all, one must focus on the composition of the peripheral blood. In 90% of patients with SLE, normochromic anemia, leukopenia and a sharp increase in ESR. In 83% of patients with SLE in urine analyzes - proteinuria of varying severity, leukocyte, erythrocyte - and cylindruria. It is characteristic that coarsely dispersed fractions prevail among urine proteins, that is, proteinuria is not selective. Detection of LE cells is considered typical. However, it has now been shown that this phenomenon is characteristic not only of lupus erythematosus and is also relatively rare. LE-cells were found in 48% of patients. Of greatest importance is the determination of antibodies to native DNA and antinuclear factor (that is, antibodies to denatured DNA in blood serum). The level of total hemolytic activity of complement in serum is reduced due to its activation by the classical pathway. Antibodies to native DNA and antinuclear factor were found in 94% of patients with SLE. Biochemical blood test: increased creatinine, ALAT, ASAT - in 25% of patients, increased creatine kinase in 28% of patients. X-ray and computed

tomography of the chest - in 38% of patients, pleural lesions (pleurisy), lupus pneumonia, pulmonary embolism.

Nuclear magnetic resonance and angiography - in 56%, CNS lesions, vasculitis are detected Echocardiography - in 78% of patients, fluid was detected in the pericardial cavity, damage to the pericardium, damage to the heart valves, etc. Ultrasound diagnostics revealed diffuse liver thickening in 60% of patients, hepatosplenomegaly in 35% of patients. As a result of research. According to analyzes and clinical examinations, skin lesions at the onset of the disease appear only in 20-25% of patients, in 60-70% of patients they appear later, in 10-15% of the skin manifestations of the disease do not occur at all. Skin changes appear on areas of the body exposed to the sun: face, neck, shoulders. Lesions have the appearance of erythema (reddish plaques with scaling), dilated capillaries along the edges, areas with excess or lack of pigment. On the face, such changes resemble the appearance of a butterfly, since the back of the nose and cheeks are affected. Hair loss (alopecia) is rare, usually affecting the temporal regions. Hair falls out in a limited area. Hypersensitivity of the skin to the sun’s rays (photosensitivity), occurs in 30-60% of patients. Redness, decreased pigmentation, malnutrition of the tissues of the lips (cheilitis). Irritability, depression - rarely.Psychoses: paranoia or hallucinations, myelopathy, neuropathy and other disorders of the formation of the sheaths of nerves (myelin), mononeuritis, polyneuritis, aseptic meningitis in 68% of patients. Clinical lesions of the digestive tract are diagnosed in 20% of patients with SLE. Lesions of the esophagus, impaired swallowing, enlargement of the esophagus occur in 5% of cases. Ulcers of the stomach and 12 intestine are caused both by the disease itself and by side effects of treatment, especially after treatment with NSAIDs and corticosteroids. All patients in the course of the process (acute, subacute, chronic) receive the following drugs: from cytostatic immuno suppressants cyclophosphamide (CF) or azathioprine, methotrexate (according to an individual dose).Patients with SLE with low disease activity are prescribed small doses of GC (prednisolone <10 mg / day), with moderate activity - medium doses (20–40 mg / day). In the presence of severe organ pathology - diffuse lesions of the central nervous system (CNS), lupus nephritis, hematological disorders (thrombocytopenia, hemolytic anemia), generalized skin lesions - higher doses of HA are needed -> 40 mg / day. Aminoquinoline derivatives (AP) occupy a special place in the treatment of SLE patients. Chloroquine phosphate and hydroxychloroquine sulfate (Plaquenil) have been successfully used for many years in SLE, mainly in cases of low to moderate disease activity. A distinctive feature of AP is the low frequency of adverse reactions requiring discontinuation of treatment, while the toxicity of hydroxychloroquine (Plaquenil) is half that of chloroquine. High hopes in SLE patients are undoubtedly associated with rituximab, which chimeric monoclonal antibodies to surface receptors of lymphocytes - CD20. TNF-α blockers

(Infliximab, Adalimumab, Etanercept). The majority of patients received monotherapy with rpituximab (4 infusions per week at a rate of 375 mg / m2), and 30% received a combination of the drug with CP. In general, rituximab treatment led to a significant decrease in disease activity in more than 80%. The clinical effect of rituximab was accompanied by a positive dynamics of morphological changes in the glomeruli according to repeated biopsies. Discussion. After long-term prescriptions of NSAIDs (dikloberl, nimesil, ortaphen, naklogen), glucocorticoids, TNF-α blockers (Infliximab, Adalimumab, Etanercept) from the gastrointestinal tract, patients complained of lack of appetite, nausea, vomiting, heartburn, pain in various parts belly.

Conclusions According to biochemical and laboratory parameters, the patients are recommended the following drugs: add (hepatoprotector of Uzbek production) 0.01 x 3 times a day for 1 month, CHOLOGENIC COLLECTION OF KHODJIMATOV (Species Cholagoge Chodjimatovi) 2 times a day, corishpan (choleretic) 1 tab 3 times a day, omez 20 mg 1 tab 2 times a day, misoprostol 400 mcg 2 times a day. After this treatment, a month later, the patients' complaints of lack of appetite, nausea, vomiting, heartburn, pain in various parts of the abdomen sharply decreased.

Литературы:

1. Якубова А.Б., Палванова У.Б. Проблемы здоровья связанные с экологией среди населения Приаралья мақола Научно-медицинский журнал "Авиценна" Выпуск №13. Кемерово 2017г.12-15 стр.

2. Якубова А.Б., Абдуллаев Р.Б., Дусчанов Б.А. /К диетотерапии больных хроническим гепатитом проживающих в экологически неблагоприятных условиях Южного Приаралья мақола Вестник межрегиональной Ассоциации «Здравоохранения Поволжья». Россия. 2003. № 9. 39-41 бет.

3. Biour M., Jaillon P. Drug-induced hepatic diseases // Pathol Biol (Paris) 1999; 47: 928-37.

4. Bjorkman D. Nonsteroidal anti-inflammatory drug associated toxicity of the liver, lower gastrointestinal tract and the esophagus // Am J Med. 1998; 105: 17-21.

5. Bueverov A.O. Medicinal lesions of the liver // breast cancer; 2001; 13-14. ru/doc

6. Denk H. Drug-induced liver injury. Verh Dtsch Ges Pathol 2002; 86: 120-5.

7. Drapkina O. M. (doctor of medical sciences, prof.), Ashikhmin Y.I. Strategies for reducing the risk of liver damage in patients receiving

8. Khazanov A.I., Rumyantsev O.N., Kalinin A.V. and other Features of medicinal and viral medicinal lesions of the liver // Klin. Bulletin. 2000; 1. nonsteroidal anti-inflammatory drugs \ Journal of Rheumatology. Traumatology. Orthopedics "No. 1 2016 No. 2

9. Lammert F., Matern S. Hepatopathien durch medikamente // Schweiz. Rundsch. Med. Prax. 1997; 16: 29-30: 1167-1171.

10. Podrezova N.V., Sapova A.I., Matvienko E.V., Razinkova N.S., Khmelevskaya \ "EFFECTIVE PHARMACOTHERAPY \ I.G. FSBEI HE" Kursk State Medical University "of the Ministry of Health of Russia, Russia, Kursk, K. Marx st., 3, tel.: +7 (4712) 588-137, e-mail: kurskmed@mail.ru, podrezovanatasha@yandex.ru,

11. Sherlock S., Douley J. Liver and biliary tract disease: practical. hands. M.: Geotar-Medicine, 1999; 864.

12. Strohmeyer G., Weik G. Lebberschadigung durch medicamente // Z. Gastroenterol. 1999; 37; 5: 367-378.

13. Vaquero J., Blei A.T. Etiology and management of fulminant hepatic failure // Curr Gastroenterol Rep. 2003; 5: 39-47