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DRUG-INDUCED AUTOIMMUNE HEPATITIS: SYSTEMATIC REVIEW AND CASE STUDY

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The authors declare that they have no conflicts of interest

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Abstract

Drug-induced liver injury (DILI) is one of the types of adverse reactions to drugs that occur as a result of their hepatotoxic effect. The pathogenesis of drug-induced autoimmune hepatitis (LIAH) is based on the production of autoantibodies to neoantigens, which are proteins of the cytochrome P450 system, which are the result of the reaction of drug metabolites. A clinically relevant problem, such as drug-induced liver damage, affects 1-1.5 million patients almost every year. The annual incidence of DILI ranges from 2.3-13.9 per 100,000 population in population studies from Europe. The Icelandic population study recorded the highest rates of 19.1 per 100,000 population per year. And in the only study based on the US population, it was found that DILI is approximately 3 per 100,000 population. Acute hepatitis is currently a well-known manifestation, and accounts for more than 90% of liver damage caused by medications. According to studies, 2.9 - 8.8% of DILI and 2 - 18% of AIH are associated with drug-induced autoimmune hepatitis. The incidence of drug-induced liver damage with the presence of antibodies (antibodies to nuclear antigen, smooth muscle and soluble liver antigen) to AIH is 83% for nitrofurantoin, 74% for minocycline, 60% for methyldopa and 43% for hydralazine.

Дәрімен шақырылған аутоиммунды гепатит: жүйелік шолу және клиникалық жағдай

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Түйінді сөздер:
дәрімен шақырылған бауырдың зақымдануы, клиникалық жағдай, RUCAM, дәрімен шақырылған аутоиммунды гепатит

Тұжырым

Дәрімен шақырылған бауырдың зақымдануы – бұл гепатотоксикалық әсерінің нәтижесінде пайда болатын дәрілік препараттардың жағымсыз реакциялардың бір түрі. Дәрімен шақырылған аутоиммунды гепатиттің патогенезінде дәрілік зат метаболиттерінің реакциясының нәтижесі болып табылатын Р450 цитохромы жүйесінің ақуыздары- неоантигендерге аутоантиденелердің өндірілуіне негізделген.

Дәрімен индуцирленген бауырдың зақымдануы, жыл сайын 1-1,5 миллион пациенттерді қамтитын клиникалық маңызды мәселе.

Бауырдың дәрі салдарынан зақымдалуының жыл сайынғы жиілігі Еуропадағы популяциялық зерттеулерде 100 000 халыққа шаққанда 2,3-13,9 санын құрайды. Исландия бойынша популяциялық зерттеу жылына 100 000 халыққа шаққанда ең жоғарғы көрсеткіш - 19,1 көрсетті. Ал АҚШ популяциясына негізделген жалғыз зерттеуде «бауырдың дәрі салдарынан зақымдалуы» шамамен 100 000 халыққа 3 көрсеткішін құрайтыны анықталды. Жедел гепатит бүгінгі таңда,дәрінің салдарынан,бауырдың зақымдалуының 90%-н құрайтындығының айқын көрінісі болып табылады. Зерттеулерге сәйкес, бауырдың дәрі салдарынан зақымдалуы 2,9-8,8% және аутоиммунды гепатит 2-18%, дәрілік аутоиммунды гепатитпен байланысты. Бауырдың дәрі әсерінен зақымдалуы антиденелердің болуының (ядролық антигенге, тегіс бұлшық етке және бауырдың еритін антигеніне) жиілігі аутоиммундық гепатитке 83%, нитрофурантоинға 74%, моноциклинге 60%, метилдопаға 43% және гидразалинге 43% құрайды.

Лекарственно-индуцированный аутоимунный гепатит: систематический обзор и клинический случай

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Аннотация

Лекарственно-индуцированное повреждение печени — это один из видов нежелательных реакций на лекарственные препараты, которые проявляются в результате их гепатотоксического влияния. В основе патогенеза лекарственно-индуцированного аутоиммунного гепатита лежит выработка аутоантител к неоантигенам, представляющим собой белки системы цитохрома Р450, которые являются результатом реакции метаболитов лекарственных средств.

Клинически актуальная проблема, как лекарственно-индуцированное поражение печени, затрагивает 1–1,5 миллиона пациентов почти каждый год.

Ежегодная заболеваемость лекарственно-индуцированным повреждением печени колеблется от 2.3-13.9 на 100 000 населения в популяционных исследованиях из Европы. В исландском популяционном исследовании были зарегистрированы самые высокие показатели 19.1 на 100 000 населения в год. А в единственном исследовании, основанном на населении США, было установлено, что лекарственно-индуцированное повреждение печени составляет примерно 3 на 100 000 населения. Острый гепатит в настоящее время является широко известным проявлением, и составляет более 90% повреждений печени, вызванных лекарствами. По данным исследований 2.9-8.8% лекарственно-индуцированного повреждения печени и 2-18% АИГ связаны с лекарственно-индуцированным аутоиммунным гепатитом. Встречаемость лекарственно-индуцированного повреждения печени с наличием антител (антитела к ядерному антигену, гладким мышцам и растворимому антигену печени) к АИГ, составляет 83% для нитрофурантоина, 74%, для миноциклина, 60% для метилдопы и 43% для гидралазина.

Introduction

The authors of the article from the Netherlands found that about 40% of people with medical drug induced liver damage had elevated levels of immunoglobulin G, in 60-70% of cases there were positive antibodies to nuclear antigen (ANA) and smooth muscle (SMA) [3]. Autoimmune hepatitis, developed as a result of drug damage to the liver, accounts for approximately 9% of all cases of AIH [3]. According to the results of a study in Colombia, drug-induced autoimmune hepatitis makes up an insignificant part of AIH [4]. Even if the clinical and histological characteristics may be similar, but patients with LIAH are more likely to suspend treatment with a low risk of relapse, progression to cirrhosis or the need for liver transplantation [4]. We studied the epidemiology of drug-induced autoimmune hepatitis in the PubMed and Science Direct databases.

Material and methods

We conducted a literature review on databases such as PubMed and Science Direct for the period from 2010 to 2022 in English by keywords: drug induced liver injury, drug induced liver injury clinical case, RUCAM, drug-induced autoimmune hepatitis. Next, filters were used by publication date, research design and access to the article. Articles with no clinical significance were excluded. A total of 29 articles were included that met the criteria.

At the second stage, the analysis of a clinical case was taken from the Central Municipal Clinical Hospital, department of gastroenterology, with an established diagnosis of drug-induced autoimmune hepatitis was carried out. A retrospective research method was used.

Results and discussion

The RUCAM scale is used to determine the causal relationship of drug-induced liver injury [8]. There are 3 main types of drug-induced liver injury based on the ratio of serum enzymes: hepatocellular, mixed and cholestatic [1].

Classification of drug-induced liver injury (DILI) according to the AASLD Practice Guide for Liver Damage Caused by Drugs, Herbs and Dietary Supplements from 2022:

By mechanism, DILI can be classified as either direct (i.e., dose-dependent, internal , and predictable) or idiosyncratic (largely dose-dependent, idiosyncratic and unpredictable) (Table 1) [9].

Table 1.
Classification of drug-induced liver injury [9]

Mechanistic classification	Direct hepatotoxicity	Idiosyncratic hepatotoxicity	Indirect hepatotoxicity
Incidence	Common	Rare	Intermediate
Dose relatedness	Yes	No	No
Predictable	Yes	No	Partially

Reproduced in animal models	Yes	No	Not usually
Latency	Rapid (days)	Variable (days to years)	Delayed (months)
Phenotypes of injury	Serum AST, ALT, or ALP elevations, hepatic necrosis, acute fatty liver, nodular regeneration	Mixed or cholestatic hepatitis, bland cholestasis, chronic hepatitis	Immune-mediated hepatitis, fatty liver, chronic hepatitis
Examples	Acetaminophen, niacin, intravenous methotrexate	Amoxicillin-clavulanate, cephalosporins, isoniazid, nitrofurantoin	Immune checkpoint inhibitors, anti-CD20 monoclonal Ab, protein kinase inhibitors
Mechanism of injury	Intrinsic hepatotoxicity that is dose-dependent	Idiosyncratic host metabolic or immune reaction	Indirect effect on liver or host immunity

The third mechanism of hepatotoxicity is called indirect drug-induced liver injury, which occurs when the biological action of the drug affects the host's immune system, which leads to a secondary form of liver damage [9]. The advantage for studying the molecular mechanisms of liver damage caused by medications and herbs is that the antigen (drug, its metabolite) is known, which remains unknown for autoimmune hepatitis [10]. In order to choose the right therapy and distinguish drug-induced autoimmune hepatitis from drug-induced liver damage without autoimmune hepatitis (AIH), it is necessary to perform a liver biopsy [7]. According to the results of a retrospective cohort study conducted from January 2010 to January 2020 among patients in one of the medical centers in China, it was found that with each relapse, the latency period decreased, and laboratory tests decreased [11]. In patients with chronic drug-induced liver injury, the risk was high after the second episode [11]. A group of persons with a possible predisposition to AIH were postmenopausal female patients with elevated levels of serum immunoglobulin IgG [11].

A study conducted at the University Hospital of Brighton and Sussex, UK, found that the natural course of drug-induced autoimmune hepatitis is similar to AIH, especially in terms of the presence of severe fibrosis on admission and the inability to maintain remission when immunosuppression is withdrawn [12]. After carefully reviewing the data at the Mayo Clinic in Rochester, Minnesota, researchers concluded that a significant proportion of patients with AIH have drug-induced AIH, mainly due to nitrofurantoin and minocycline [13]. Department of Gastroenterology and Hepatology, Faculty of Medicine, Mie University, Japan, studied seven cases of autoimmune hepatitis that developed after drug-induced liver injury, in which it was found that a wide range of drugs can cause AIH [14]. Given that AIH can develop even after normalization of liver enzymes, careful follow-up is required in all cases of drug-induced liver injury [14].

Based on the results obtained in the Department of Surgical Gastroenterology in China, it should be noted that the differences in ALT, AST and CD4 + Foxp3 + CD25 - Treg between patients with drug-induced autoimmune hepatitis and patients with AIH are clinically useful for differentiating these two diseases on their early stage [15].

A study conducted in Japan revealed clinical characteristics of drug-induced liver injury, which showed histological findings similar to AIH [16]. In such patients, a liver biopsy is recommended to determine the appropriate treatment tactics [16]. A study conducted at the Department of Anesthesiology and Critical Care at Johns Hopkins University showed that drug-induced autoimmune hepatitis is the most common process of drug-induced liver hypersensitization, which is observed in approximately 9–12% of patients with autoimmune hepatitis [17]. According to an article published in the Department of Gastroenterology, Kyoto Okamoto Memorial Hospital (Japan), a clinical case of a patient diagnosed with IgG4-associated AIH with an etiology presumably caused by drugs is presented [18]. Oral prednisolone was started and discontinued after achieving biochemical remission. Autoimmune hepatitis recurred after discontinuation of steroids; however, remission was achieved with ursodeoxycholic acid [18].

According to a search on the Medline database, since 1966 there are 14 registered cases of AIH caused by statins [19]. The article by E. Kawasaki et al. describes 2 clinical cases, which report on patients with type 1 diabetes who developed autoimmune hepatitis (AIH) after taking statins [19]. Most cases of AIH were diagnosed within 1 year of statin use, with a mean age of 56.7 ± 11.0 years [19]. The article by A. Villamil and others reported 2 cases of acute autoimmune hepatitis in patients with multiple sclerosis treated with IFN-beta 1a [20]. One of the complications of alpha-IFN treatment in patients with chronic viral hepatitis is the development of autoimmune hepatitis [20]. An article published in Canada found that transient liver enzyme disorders are relatively common in children receiving anti-TNF treatment [21]. Anti-TNF-associated drug-induced liver injury with autoimmune features is rare but must be recognized before therapy can be discontinued [21]. A study in Iceland showed that when assessing clinical use and safety risk, TNF-α inhibitors were more likely to cause liver damage compared to other biologics [22]. A study conducted at the Walter Reed National Military Medical Center (USA) showed a case of infliximab-induced seronegative AIH responding to budesonide therapy, with a successful change in the treatment regimen for inflammatory bowel disease to vedolizumab [23]. A review conducted at the Institute for Liver Research, King's College Hospital, London (UK) proposes a structured practical approach to the diagnosis and treatment of a group of patients with autoimmune hepatitis [24]. In studies conducted at Haset Tepe University, Department of Gastroenterology, in Turkey, it was shown that after the use of tumor necrosis

factor-α (anti-TNF-α) blockers, including infliximab, etanercept and adalimumab, mild to moderate increased activity of liver enzymes, cases of severe hepatitis were rare [25]. For this reason, TNF-α blockers are considered as a potential cause of drug-induced autoimmune hepatitis [25]. According to a review conducted in the Department of Gastroenterology, Belgrade Children's University Hospital, immune-mediated hepatotoxicity of albendazole was found to be one of the possible mechanisms of liver damage [26]. The use of albendazole in the treatment of parasitic infections, especially in children, requires careful monitoring [26]. This article, conducted at the Department of Medicine and Pediatrics, Rush University Medical Center, Chicago (USA), describes a clinical case of a male adolescent who developed autoimmune hepatitis induced by minocycline [27]. A study by the Department of Gastroenterology and Hepatology, Rutgers School of Medicine, New Jersey, showed that drug-induced liver injury remains the most common cause of acute liver injury in the United States [28]. Drug-induced liver injury is one of the most challenging diseases faced by hepatologists due to the variety of drugs used in clinical practice, available herbs and nutritional supplements with hepatotoxic potential, the ability of the disease to present with a variety of clinical and pathological phenotypes, and also due to the current lack of specific biomarkers [29].

Case study

Patient K., 52 years old, complained about severe weakness, headaches, dizziness, shortness of breath during physical activity, fatigue. From the anamnesis of the disease: she has been ill for 2 years the history of disease about 2 years, when AIH was first diagnosed. Concomitant diseases: Autoimmune thyroiditis. She took dietary supplements for a long time. The diagnosis was made. The diagnosis is: Drug-induced liver injury, mixed variant (requires dynamic monitoring for possible drug-induced autoimmune hepatitis) with pronounced biochemical activity at the onset of the disease (126 ULN ALT dated 11.04.2020) and moderate biochemical activity at the time of examination (5.9 ULN ALT) (Figure 2, 3, 4). Autoimmune thyroiditis, subclinical hypothyroidism. Dyslipidemia. The results of laboratory studies are presented in Figures 1, 2, 3, 4 5.

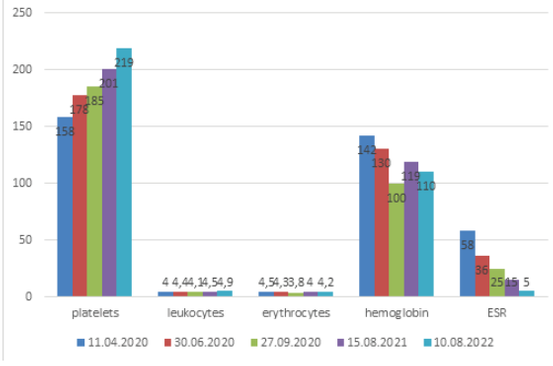


Figure 1.
Results of Complete blood count

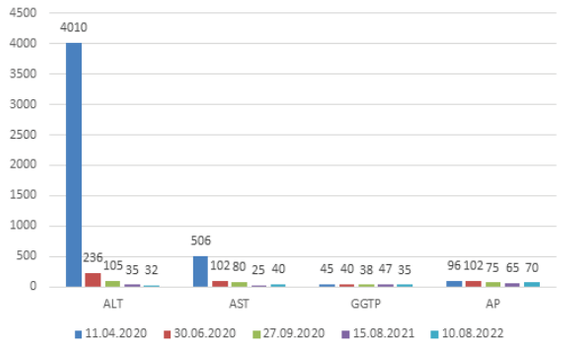


Figure 2.
Results of a biochemical blood test

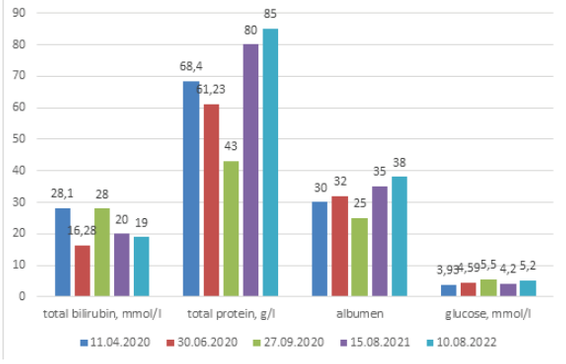


Figure 3.
Results of a biochemical blood test

Figure 4.
Results of a biochemical blood test

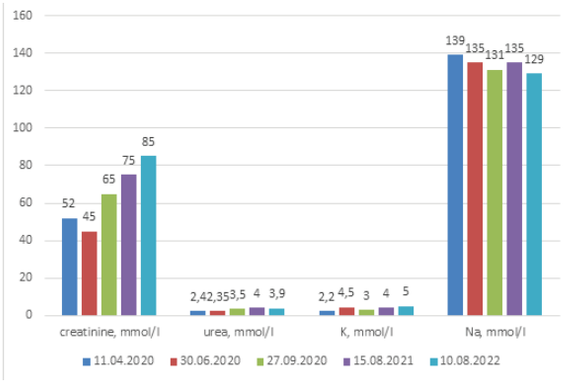
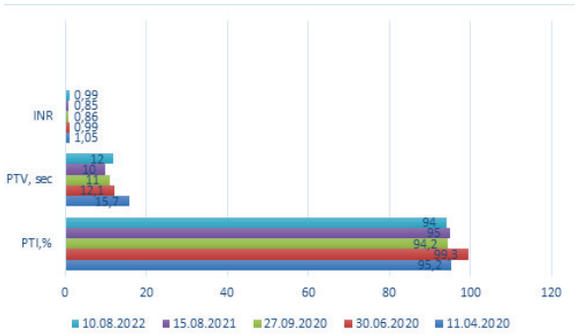


Figure 5.
Coagulation test



The patient was prescribed standard immunosuppressive therapy with Prednisolone 60 mg in 2-3 times a daily, Ursodeoxycholic acid 500 mg 2 times a day before breakfast and at night for a long time. When re-examined about 3 months after, there was a biochemical response to immunosuppressive therapy (minimum biochemical activity). In this regard, it was recommended to reduce the dose of Prednisolone from 20 mg to 5 mg per week to 10 mg / day orally daily until 11 am - for a long time; alternative option - Budesonide (Budenofalk) 3 mg 3 times a day before meals - long-term. Azathioprine or 6-mercaptopurine 50 mg/day. At the moment, the patient is under our careful dynamic observation.

Conclusions

Drug-induced liver injury and drug-induced autoimmune hepatitis may be similar in clinical laboratory findings. The final role in the differential diagnosis is played by a liver biopsy, which is necessary for further treatment.

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