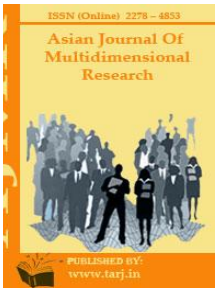
AJMR

ISSN: 2278-4853 Vol 9, Issue 10, October, 2020 Impact Factor: SJIF 2020 = 6.882



Published by: TRANS Asian Research Journals

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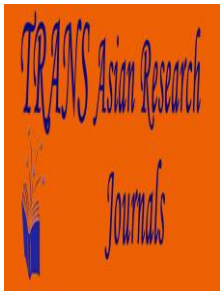
Asian Journal of Multidimensional Research

(A Double Blind Refereed & Peer Reviewed International Journal)



DOI: 10.5958/2278-4853.2020.00271.2

CLINICAL COURSE OF CHRONIC VIRAL HEPATITIS C IN CHILDREN

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ABSTRACT

In the following article clinical course processes of chronical hepatitis C in children is analysed. The incidence of seropositivity is identified. The authors have analysed the arginase activity of saliva which maintained the indices of chronical hepatitis. The existence of a hematosalivation barrier in the body, the presence of a metabolism between blood and saliva, as well as the homeostatic role of these biological fluids for each other - all these facts make saliva capable in some cases of replacing blood in laboratory tests. Thus, the accumulated literature data indicate that the chronic hepatitis C virus may remain asymptomatic for a long time, but a number of extrahepatic manifestations in the oral cavity can be detected in patients.

KEYWORDS: *Chronic Viral Hepatitis, Public Health Problem, Liver Dysfunction, Sjorgen's Syndrome, Precancerous Conditions, Dysbiotic Processes*

INTRODUCTION

Viral hepatitis C (HCV) is currently one of the urgent public health problems due to its prevalence in the population, the high incidence of liver cirrhosis and hepatocellular carcinoma, the development of extrahepatic manifestations that determine the difficulties in diagnosing the disease and its treatment. According to WHO estimates, in 2015, 1.75 million new cases of HCV infection were recorded worldwide (23.7 new cases per 100,000 people). WHO estimates that approximately 399,000 people died from hepatitis C in 2016, mainly from liver cirrhosis and hepatocellular carcinoma (WHO Newsletter, 2019). Hepatitis C virus causes 20% of all cases of acute hepatitis, and chronic HCV infection is responsible for the development of 70% of cases of chronic hepatitis, 40% of all cases of terminal cirrhosis of the liver, 60% of hepatocellular carcinoma and 30% of the causes the patient is referred for liver transplantation [9, 10]. Of interest is the fact that infection with hepatitis viruses, including HCV, is also possible through the oral mucosa, for example, with kisses. It has already been proven that hepatitis B and C viruses are transmitted by all body fluids - up to 30% hepatitis B virus and up to 5% hepatitis C virus [29].

Salivary transmission of hepatitis B and C viruses can be one of the non-parenteral modes of transmission [19]. Of particular interest is the presence of the virus in the saliva of patients with chronic viral hepatitis C (HVHS).

To date, a large number of studies have already been accumulated devoted to studying the amount of hepatitis C virus RNA in the saliva of patients with chronic hepatitis C (HCV) [17, 28].

Liver dysfunction can manifest itself with various changes in the oral cavity, namely, in the form of ictericity of the mucous membrane, bleeding disorders, the presence of petechiae, a tendency to hematomas, gingivitis, gingival bleeding (even in response to minimal trauma), hepatic odor from the mouth, cheilitis, smooth and atrophic tongue, xerostomia, perioral rash, soreness of the oral cavity [30].

According to various authors, patients with hepatitis C are prone to tooth decay, suffer from a loss of self-esteem due to poor aesthetics of the oral cavity, and sometimes experience difficulties with nutrition due to an unsatisfactory state of the oral cavity. All this leads to a decrease in the quality of life [22].

In a study by Coates E.A. et al. (2000) studied the state of the oral cavity in patients with chronic hepatitis C compared with patients without HCV markers. Patients were between 25 and 50 years old. Although there were no significant differences between the groups, nevertheless, HCV-infected patients were more likely to have problems associated with the state of periodontal structures. So, in half of patients with HCV there was a decrease in the amount of saliva secreted. 71% of patients with HCV showed a decrease in quality of life due to the presence of significant pain in the oral cavity [22].

In recent years, the relationship between HCV and the development of lichen planus, which also affects the mucous membrane of the oral cavity, has been proven, and the frequency of occurrence varies depending on the geographical region. So, the most typical development of this pathology is for the South European region and Japan [18, 20].

Given that seropositivity for anti-HCV in patients with lichen planus is much higher, for example, in Israel, all patients with lichen are regularly tested for antibodies to HCV [33].

HCV is known to affect the salivary glands, but the exact nature of this effect remains to be fully understood. It is believed that hepatitis C virus causes shergen-like syndrome. There are suggestions that HCV is the cause of Sjogren’s syndrome, but this is controversial. It is unclear whether the virus can cause a similar pathological condition; primary Sjogren's syndrome or HCV is directly responsible for the development of Sjogren's syndrome in a specific subgroup of patients [21].

Increased prevalence of HCV infection in patients with squamous cell carcinoma of the oral cavity was discovered back in 1995 by Nagao Y. and coauthor [26]. A 2004 study in the United States showed that 21% of 99 patients with squamous cell carcinoma had HCV markers [27].

It has been shown that HCV does not affect the survival of such patients [24].

In another study from Japan [32], the authors found an increase in the incidence of HCV in patients with oral cancer, but this difference was not significant after the data were adjusted for age.

However, HCV is a common cause of cirrhosis, which, in turn, can be an independent risk factor for oral cancer [31].

On the other hand, it has been shown that precancerous conditions such as leukoplakia and oral epithelial dysplasia are not associated with HCV infection [20, 25].

Special attention should be paid to the issues of periodontal tissue lesions in HCV.

The pathogenesis of periodontal diseases is based on serious violations of microbiocenosis in combination with dysfunction of the body's immune system against the background of generalized inflammatory, dystrophic and vascular changes [5].

It is known that the development of periodontal pathology is influenced by concomitant chronic diseases of the liver and hepatobiliary system, which currently have a predominantly viral nature [2, 3, 7].

According to a study by Farghaly A.G. et al. (1998), cases of periodontal disease were noted more often in children with hepatitis markers than in the group of patients in whom hepatitis markers were not detected. At the same time, hepatitis C markers were detected more often than hepatitis B markers (26% and 13% versus 22% and 8%, respectively). Also, in patients with periodontal disease, unstimulated saliva showed a higher degree of detection of HBsAg, anti HBc, anti-HCV or both anti-HCV and / or anti-HBc than in the control group (100% versus 66.7%, 50% against 23.5%, 23.1% against 0.0% and 42.3% against 18.2%, respectively) [23].

As one of the pathogenetic factors that can induce numerous clinical and laboratory phenomena described in CVHB and CVHV, a number of researchers consider endotoxin of gram-negative bacteria, causing the development of a complex of pathophysiological, immunological and other biological processes [6, 11, 12].

The main anti-endotoxin barrier is the liver [2, 6].

With the development of dysbiotic processes in the oral cavity, as well as with other pathological processes in the gastrointestinal tract, the intake of endotoxin into the systemic circulation increases, which can cause an increase in intoxication syndrome and aggravation of chronic inflammation in the tissues [6, 11].

In his research, Fazylova Yu.V. showed that the level of specific antiendotoxin antibodies in patients with CVD against CVHB and CVHC is an integral indicator of immunological deficiency and indicates the development of systemic endotoxemia as a result of microbial aggression associated with the severity and prevalence of periodontal disease and activity of chronic viral hepatitis [14].

Of particular scientific interest is the study conducted by S. Kolesov. et al. [8], dedicated to the study of salivary arginase activity in children with HVHV and KhVGS.

As you know, arginase catalyzes the hydrolysis of arginine with the formation of ornithine and urea. Since disruption of urea synthesis leads to an increase in the amount of ammonia in tissues, it is generally accepted that the level of arginase activity reflects the degree of detoxifying liver function [4, 15].

In addition, there is evidence that in clinical studies, the determination of arginase in blood serum can be used as a specific marker that allows you to detect liver damage at an earlier stage

than in the study of aminotransferases, which is important for the correct treatment of liver diseases including chronic hepatitis.

It should be especially noted that at present, an increase in researchers' interest in arginase can be expected, since it is proved that this enzyme can act as a limiting factor in the formation of nitric oxide in the body, a unique messenger whose biological role is very large: the physiological effect of NO varies from modulation of the vascular system to the regulation of immune processes and control of neuronal functions [1].

In addition to the liver, arginase activity was also found in other organs, tissues and substrates of the body, including saliva [16]. The existence of a hematosalivation barrier in the body, the presence of a metabolism between blood and saliva, as well as the homeostatic role of these biological fluids for each other - all these facts make saliva capable in some cases of replacing blood in laboratory tests. Especially attractive is the use of saliva for pediatric practice, since obtaining oral secretions is accessible and non-traumatic.

According to the results obtained by the authors, the average values of arginase I (liver arginase) in the blood serum of children with chronic viral hepatitis B and chronic viral hepatitis C were added 8.01 and 10.35 ng / ml, respectively. The average values of the arginase activity of saliva in the group of children with chronic viral hepatitis B turned out to be two times higher than the level of this indicator in patients with chronic viral hepatitis C and amounted to 82.34 and 33.24 mmol / hr / ml, respectively. The authors concluded that the arginase activity of saliva is due to the enzymatic activity of the arginase isoenzyme from the salivary glands and is not related to the activity of arginase I (liver arginase). The decrease in the arginase activity of saliva in children with chronic viral hepatitis C, apparently, is an extrahepatic manifestation of chronic viral hepatitis C, and is characterized by changes in the dental status characteristic of this disease.

It is known that half of patients with chronic hepatitis C have extrahepatic manifestations of the disease, including damage to the oral cavity: they have a high incidence of symptoms of periodontal disease, there has been a periodontal disease and gingivitis (and in patients with chronic hepatitis C, these diseases represented mainly by generalized forms of severe and moderate) [13], lymphocytic sialadenitis.

The authors of the study believe that the need to combat infection is a biochemical mechanism that ensures a decrease in salivary arginase activity during periodontal lesions of the oral cavity. According to literature [1], arginine, not utilized by arginase, is used for the synthesis of nitric oxide, one of the functions of which is both direct participation in the fight against infection and stimulation of the immune response to it. Due to this, a peculiar dynamic relationship between aggression factors (oral infection) and the body's defensive reactions is established in the patient’s body.

Thus, the accumulated literature data indicate that the chronic hepatitis C virus may remain asymptomatic for a long time, but a number of extrahepatic manifestations in the oral cavity can be detected in patients. In this regard, the dentist plays an active role in detecting HCV infection. Further research is needed to identify a possible relationship between oral pathology and HCV.

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