

Extrahepatic manifestations of hepatitis C virus infection in the oral cavity

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Medical Studies 2025; 41 (3): 172–178

DOI: <https://doi.org/10.5114/ms.2025.154759>

Key words: hepatitis C virus, chronic hepatitis C, extrahepatic manifestations, lichen planus, xerostomia, periodontal disease.

Abstract

According to data from the World Health Organization (WHO), the number of people actively infected with hepatitis C virus (HCV) in the world currently reaches 50 million. In the last few years, there has been a revolution in the treatment of chronic hepatitis C (CHC) and now safe interferon-free regimens based on drugs with direct-acting antivirals (DAA) are available in the treatment of HCV infections, the effectiveness of which exceeds 95%. As a result, according to WHO forecasts, it is possible to eliminate HCV infection by 2030. A necessary condition to achieve this goal is the diagnosis of the disease in patients unaware of the infection. Chronic HCV infection can lead to extrahepatic manifestations, including changes in the oral mucosa such as xerostomia, lichen planus, or periodontal diseases. The aim of this publication is to draw attention to the extrahepatic symptoms with which a patient infected with HCV may present to a dentist.

Introduction

Hepatitis C virus (HCV) belongs to the genus *Hepacivirus*, family *Flaviviridae* [1].

HCV infection is transmitted by the blood-borne route, through direct contact with the blood of an infected person or indirectly through contact with instruments contaminated with the patient's blood (medical procedures: surgery, endoscopy, dental procedures, dialysis, and non-medical: tattooing, piercing, intravenous drug use). In the case of blood transfusions, the risk of transmission applies to events that occurred before 1992, when tests detecting anti-HCV antibodies became widely used in blood donation [2]. The risk of hepatitis C virus infection by the vertical route is about 5%, for the sexual transmission in a monogamous relationship is less than 1%, and increases in the case of promiscuity and coexisting sexually transmitted diseases, especially HIV infection [3].

Chronic hepatitis C (CHC) is usually asymptomatic or with uncharacteristic symptoms such as weakness, fatigue, abdominal discomfort, so it is often detected incidentally. It has been a very significant global health problem for many years. According to WHO data, the number of actively infected people is currently 50 million, and nearly 242,000 of them die annually as a result of the most serious complications

of the disease, i.e. cirrhosis and hepatocellular carcinoma [4, 5]. According to the Polish Group of Experts (PGE) on HCV, the number of infected people in Poland reaches 140,000, and only 20% of them are aware of it [6]. Diagnostics is based on serological tests to detect anti-HCV antibodies, the presence of which indicates contact with the virus, but is not evidence of ongoing infection. Only the detection of the genetic material of the virus in the blood by the polymerase chain reaction confirms active HCV infection.

Historically, we have had regimens containing interferon (IFN) in the form of subcutaneous injections combined with ribavirin to treat CHC; in a typical Polish GT1b-infected patient, treatment lasted 48 weeks, with an effectiveness of about 50%. In 2013, sofosbuvir, the first oral direct-acting antiviral (DAA) drug that blocks the virus' life cycle enzymes, was registered. The introduction of interferon-free regimens containing a combination of these new drugs started a revolution in the treatment of CHC, significantly shortening the duration of treatment (in most patients it is now 8 or 12 weeks, in exceptional cases, 24 weeks) and significantly increasing its effectiveness to over 95% [7].

The sensational effectiveness and very good safety profile have enabled causal treatment of many patients who previously could not be treated with IFN- α due to contraindications and expected side effects

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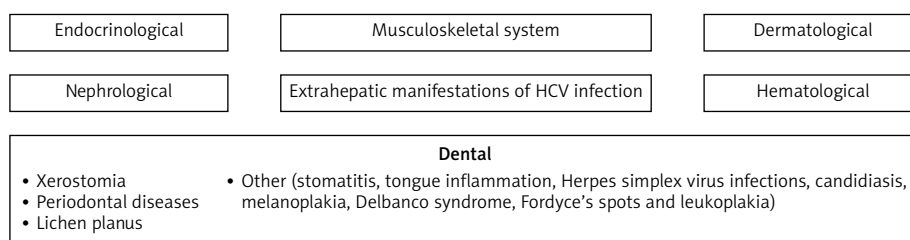


Figure 1. Extrahepatic manifestations of hepatitis C virus (HCV) infection

[7, 8]. This includes patients with comorbid neurological and psychiatric diseases, autoimmune diseases, severe cardiovascular, respiratory and renal diseases, as well as patients with advanced liver disease [9–11].

The availability of safe and effective therapy has led the WHO to set a goal of eliminating HCV as a significant public threat by 2030. Treating twelve thousand patients per year in Poland for the next decade, as planned in 2017, would completely meet the goals of the WHO strategy [12]. However, without population-based screening, the possibility of achieving this goal will remain out of reach [13]. A recent study assessing the progress of 45 high-income countries found that some, such as Japan and Iceland, could achieve full elimination with the right conceptual framework combining factors such as political will, national screening and awareness programs [12]. In Poland, thanks to recent changes in the primary care package, family physicians can play a major role in detecting the disease by referring patients under their care for anti-HCV antibody testing. Diagnostic vigilance by other medical specialists is also expected. The morbidity associated with chronic HCV infection has a broader dimension than just hepatic due to extrahepatic manifestations of CHC involving various systems and organs. The mechanisms underlying this phenomenon are both indirect – such as viral lymphotropism, complex interactions with the host immune system, and the ability to induce autoimmune reactions – and direct, related to the ability of HCV to replicate in cells and tissues other than the liver. The first extrahepatic syndrome to receive attention in the context of a pathogenetic link to HCV infection was mixed cryoglobulinemia (MC), which is also the most common extrahepatic pathology found in CHC. Other extrahepatic manifestations etiologically associated with HCV infection include: hematological disorders, diseases of the skin, kidneys, salivary glands, eyes, connective tissue, nervous system, endocrine disorders, cardiovascular disease, isolated autoimmune reactions, metabolic disorders, and mental health pathologies [14]. As a result, a patient chronically infected with HCV may visit many different specialists, including the dentist (Figure 1).

The aim of this publication is to draw the attention of dentists to how important it is for the diagnosis and treatment of HCV infection to properly assess

oral health during a routine dental examination. For the sake of their own safety and that of uninfected patients, the principles of asepsis and antisepsis should be followed, treating each patient as potentially infected.

Characteristics of the basic oral examination

Oral pathology can involve the mucosa, teeth and periodontium. Diseases of the oral mucosa constitute a comprehensive chapter of conservative dentistry, important not only for dentists, but also for doctors of other specialties.

The oral mucosa often reflects the general condition of the body. Pathological changes within it can be one of the first signs of acute and chronic diseases affecting other organs and systems. The dentist may therefore be the first person to notice the presence of oral lesions in a patient unknowingly infected with chronic hepatitis C. This will allow the correct diagnosis of HCV infection and, if the disease is diagnosed, the implementation of effective antiviral therapy.

It is crucial to conduct differential diagnostics for dental manifestations of HCV infection (Table 1).

The relationship between oral mucosal diseases and HCV infection has received increasing attention in the literature in recent years; in some people, the causal relationship is definitively established, in others it is probable or postulated [15–17].

Extrahepatic manifestations of CHC that the dentist may encounter

Xerostomia

Xerostomia, or dry mouth syndrome resulting from Sjögren-like sialadenitis (S-IS), is an extrahepatic manifestation strongly associated with HCV [18]. The first study describing the association between salivary gland disorders and CHC was published in 1992 and identified characteristic histologic changes in the salivary glands in 57% of HCV-infected patients [19]. The coexistence of lymphocytic salivary gland inflammation with CHC, according to reports by various authors, is observed in 4% to 57% of patients, and as many as 80% of HCV-infected individuals may show abnormalities in saliva or tear composition [18, 20]. Dysfunction of the salivary glands, combined with decreased salivary secretion, may resemble

Table 1. The differential diagnostics of dental manifestations of hepatitis C virus infection

Extrahepatic manifestations in the oral cavity	Other reasons of disorders
Xerostomia	Primary Sjögren syndrome
	Dehydration
	Mouth breathing
	Sialadenitis
	Sialolithiasis
	Diabetes mellitus
	Hypothyroidism
	Sarcoidosis
	Amyloidosis
	Granulomatosis with polyangiitis
	Drug-induced sicca syndrome (anti-cholinergic, anti-depressant, anti-psychotic, diuretic, anti-hypertensive, sedative, muscle relaxant, analgesic, and anti-histaminic agents)
	Head and neck radiation therapy
	Chemotherapy drugs
	Other viral infections
	Idiopathic sicca syndrome
Oral lichen planus	Trauma
	Diabetes mellitus
	Hypothyroidism
	Graft versus host disease
	Allergic reaction to dental material
	Lichen planus pemphigoides
	Chronic ulcerative stomatitis
	Lupus erythematosus
	Medications: β -blockers, angiotensin-converting-enzyme inhibitors, diuretics, oral hypoglycemics, non-steroidal anti-inflammatory drugs, penicillamine
	Idiopathic oral lichen planus
Periodontal diseases	Poor oral hygiene
	Obesity
	Diabetes mellitus
	Insulin resistance
	Type 2 diabetes
	Dyslipidemia
	Ventilator-associated pneumonia
	Alzheimer's disease
	Rheumatoid arthritis
	Osteoporosis
	Chronic kidney disease
	Chronic liver disease
	Viruses (e.g., herpes herpesviruses, SARS-CoV-2)

the changes seen in classic Sjögren's syndrome. However, despite similar clinical manifestations, the etiology of lymphocytic salivary gland inflammation in chronic HCV infection is likely different, as is the nature of salivary gland damage. Antibodies to Ro (SSA) and La (SSB) nuclear antigens, the presence of which is typical of classic Sjögren's syndrome, are observed much less frequently. There is no higher prevalence in women and more frequent association with HLA-DQB1*02 than HLA-DR3 is noted, histopathological changes are less pronounced, and lymphocytic inflammatory infiltrates are localized more perivascularly than perineurally. There is no destruction of the salivary ducts and there is a predominance of CD8 over CD4 responses [21]. HCV RNA is detected in the salivary glands, suggesting a direct cytopathic effect of the virus. However, some authors question this pathogenetic mechanism, claiming that the inflammatory process in the salivary glands is not directed against HCV, but against salivary gland proteins similar to the viral envelope proteins, based on molecular mimicry [22]. In HCV-positive patients diagnosed with Sjögren's syndrome, cryoglobulinemia, the most common extrahepatic manifestation with an established strong pathogenetic link to HCV, is significantly more common in HCV-positive patients compared to patients with Sjögren's syndrome who are not infected with the virus [23].

A patient with dry mouth poses a serious diagnostic and therapeutic challenge for the dentist. Due to reduced saliva production, increased susceptibility to caries, inflammatory mucosal changes and periodontal disease may occur. Finally identifying the cause of the existing problem is possible only in a minority of patients. Therefore, symptomatic treatment is most often used, and the possibility of effective causal treatment exists only in drug-induced xerostomia [24]. When a dentist observes no improvement in a patient with dry mouth syndrome despite ongoing therapy, it is advisable to consider referring the patient for testing for HCV infection. This step helps determine whether dry mouth is related to HCV in this particular case. Such an association is highly likely when xerostomia is accompanied by yellowing of the oral mucosa, bleeding gums, concomitant petechiae, smoothing lesions on the tongue and a drying rash at the corners of the mouth [25]. Correct diagnosis and initiation of antiviral treatment can then improve the outcome of dental interventions.

Lichen planus

Lichen planus is a chronic inflammatory disease that affects the skin and mucous membranes, including the oral mucosa, referred to as oral lichen planus (OLP). The association of lichen planus with HCV infection was first described in 1991 by Mokni *et al.* and has since been confirmed by many authors [26, 27].



Figure 2. Wilson's lichen planus – erosive form, on the cheek mucosa in a 52-year-old woman treated for chronic hepatitis C (CHC)

OLP mainly affects middle-aged adults of both sexes, with a slight female predominance [15].

The lesions usually appear on the buccal mucosa, mainly in the posterior region, sometimes extending to the corners of the mouth, and show a symmetrical distribution. Sometimes similar lesions may also appear on the tongue in the form of tree-like opacities. OLP can present in several clinical subtypes, including reticular, atrophic, discoid and erosive forms (Figures 2, 3). The most commonly observed manifestation is Wickham's striae, characterized by white papules that merge in lines to form a characteristic pattern resembling a milky white grid. The erosive form is less common [28]. It is not uncommon for multiple forms of the disease to coexist in a single patient. In the general population, the incidence of OLP is 1–2%, while it is five times higher in the chronic HCV-infected population [15]. The pathomechanism of OLP includes HCV-induced autoimmune disorders involving cytotoxic lymphocytes. In addition, potential replication of HCV in oral epithelial cells is indicated, which may directly contribute to mucosal changes [15, 29]. OLP has the potential to negatively impact quality of life due to pain associated with food intake.

Diagnosis is usually made on the basis of characteristic oral mucosal lesions; however, histopathological examination is recommended for diagnostically equivocal lesions. Differentiation should include conditions such as leukoplakia, candidiasis, lichen-like rash and pemphigoid. OLP is considered a precancerous condition, and studies indicate that 1% of patients with OLP develop squamous cell carcinoma (SCC) [30]. The established pathogenetic link between OLP and HCV underscores the importance of testing for anti-HCV antibodies in patients diagnosed with OLP during dental examinations. As early as in mid-1990s, an increased incidence of oral SCC was reported in patients with chronic HCV infection. Cancer risk



Figure 3. Wilson's lichen planus – reticular form, on the cheek mucosa in a 58-year-old woman treated for chronic hepatitis C (CHC)

was found to be unrelated to behavioral factors such as smoking or alcohol abuse. Chronic HCV infection and cirrhosis have been identified as independent risk factors for oral cancer [28]. Evaluation of the pathogenetic link between oral SCC and HCV requires further controlled studies. Nevertheless, when a dentist diagnoses oral mucosal SCC, the patient should be referred for HCV testing in addition to receiving appropriate oncological care.

Periodontal diseases

Periodontal diseases pose a major dental challenge due to their multi-causal etiopathogenesis and diverse clinical manifestations. The inflammatory process can occur within individual teeth or involve entire dental arches. The wide range of symptoms includes reddening, swelling and bleeding of the gums, even after minor trauma, loss of tissue tone, pathological tooth mobility, discharge from periodontal pockets and progressive gingival atrophy. Exposed tooth roots, often covered with plaque and tartar, increase susceptibility to caries.

There are various classifications of periodontal disease, based on clinical symptoms and etiological factors. Due to the fact that inflammatory lesions in

the periodontium can be associated with concurrent systemic diseases, the term “gingivitis modified by systemic factors” is encountered in periodontal nomenclature [31]. Epidemiological studies indicate a higher prevalence of periodontal disease in patients with chronic HCV infection [32]. Coates *et al.* examined the oral health of 87 individuals aged 35–44 with CHC, comparing this group to the general population [33]. Oral changes identified by the Caries Severity Index, which assesses the number of decayed teeth, cavities and fillings, were three times more prominent in HCV-infected patients, indicating more decayed teeth and cavities but fewer fillings compared to the control group. Evaluation of periodontal status using the Community Periodontal Index of Treatment Needs showed increased gingival bleeding and deepening of gingival pockets in patients with chronic HCV [34]. It has been postulated that the increased risk of developing periodontal disease in patients with CHC is associated with a combination of factors, including immune dysfunction, chronic inflammation, insulin resistance and inadequate dental care. Unlike lymphocytic parotitis, OLP and oral SCC, where a direct link to HCV has been documented, periodontal disease, based on the available literature, appears to be an indirect consequence of chronic HCV infection.

Other diseases

Extrahepatic oral manifestations suspected to be related to HCV infection are receiving increasing attention in the literature. These symptoms include lesions such as stomatitis and tongue inflammation, Herpes simplex virus infections, oral candidiasis, melanoplakia, Delbanco syndrome, Fordyce spots and leukoplakia [35, 36]. However, there is a lack of literature data confirming the association of burning mouth syndrome and chronic recurrent aphthae with HCV infection [37].

Conclusions

Advances in the treatment of patients with chronic hepatitis C have made the elimination of HCV infection a real possibility in the coming years. Achieving this goal requires collaboration with health care professionals from various specialties, including dentists, to identify patients who may be unaware of infection. Knowledge of the risk factors, methods of transmission and extrahepatic manifestations of CHC that dentists may encounter is crucial to their contribution to this collaborative effort. Due to the presence of extrahepatic manifestations of HCV infection in the stomatognathic system, it is crucial that dentists perform a comprehensive evaluation of oral pathology and detailed differential diagnosis of lesions during routine examinations. Careful observation and thorough evaluation of pathological changes can enable early diagnosis and implementation of appropri-

ate treatment in HCV-infected patients who may not have been aware of the disease.

Funding

The science project of Jan Kochanowski University in Kielce, Poland (project number SUPB.RN.23.011).

Ethical approval

Not applicable.

Conflict of interest

The authors declare no conflict of interest.

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Received: 15.12.2023

Accepted: 1.10.2024

Online publication: 30.09.2025