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REVIEW

The Significant of the Oncoviruses in Saliva Patients in Dental Clinics

Nasrin Mohamadi[1], Manoochehr Makvandi[2], Faramarz Zakavi[3]

[1]Postgraduate Student, Department of Restorative Dentistry, School of Dentistry, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran. [2]Virology Department, School of Medicine, Jundishapur University of Medical Sciences, Ahvaz, Iran. [3]Associate Professor, Department of Restorative Dentistry, School of Dentistry, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran.

Abstract

Saliva possesses the potential to contain numerous pathogenic viruses, which constitutes a significant concern, particularly within dental clinics. The viruses identified encompass both low-risk and high-risk human papillomavirus, herpes viruses such as HSV1 and HSV2, human varicella zoster virus, Epstein-Barr virus, human cytomegalovirus, HHV6, HHV7, HHV8, hepatitis A virus, hepatitis B virus, hepatitis E virus, rabies virus, JC virus, BK virus, and influenza A and B viruses, alongside coronaviruses. These viral entities are responsible for a substantial incidence of morbidity and mortality on a global scale. Despite the absence of definitive therapeutic interventions for the majority of these infectious viruses, there exist limited vaccination programs targeting several of them, including both low-risk and high-risk human papillomavirus, hepatitis B vaccine, influenza A and B vaccines, and the COVID-19 vaccine. It appears imperative that enhanced attention and preventive strategies be instituted in dental clinics to mitigate the transmission of infectious diseases propagated through saliva.

Keywords: Oncoviruse- saliva- dental clinic

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Introduction

Viruses can be transmitted via several routs to human body and cause certain type of infection, however the transmission of viruses via contaminated saliva constitutes a significant concern, particularly within dental clinics.

It has also been posited that both benign and malignant orofacial neoplasms may have associations with Human Papillomavirus (HPV) and Herpesviruses [1, 2].

Human papillomavirus

Human Papillomaviruses (HPV) have double stranded DNA molecule of about 8000 base pair (bp) [1]. Human Papillomaviruses comprise over 200 distinct genotypes, which are classified into categories of low- and high-oncogenic risk types [1]. Low-risk (LR) types (LR-HPV6, 11, 34, 40, 42, 43, 44) predominantly present as benign genital warts, clinically referred to as condyloma, as well as laryngeal papilloma [3, 4]. In contrast, high-risk (HR) types, particularly HR-HPV16 and HR-HPV18, are etiologically linked to cervical cancer, vulvar, vaginal, anal, penile and oropharynx carcinomas [1], whereas other high-risk (HR) types such as 26, 30, 34, 53, 66, 67, 69, 70, 73, 82, 85, and 97 have been reported with low prevalence in cervical cancer cases [5]. The HPV family has been demonstrated to possess the capability to infect and transform epithelial cells [6]. Numerous investigations have documented the presence of human papillomavirus in the saliva of individuals diagnosed with oral cancer [7-12]. Additionally, the identification of human papillomavirus has been reported in dental clinical settings [13-15]. The E6 and E7 oncoproteins of human papillomaviruses exert critical influence in the pathogenesis of cancer by interfering with cell cycle regulation and inhibiting programmed cell death [16]. The E6 oncoprotein has the capacity to specifically interact with the cellular p53 protein, resulting in its degradation and subsequent reduction in concentration within cancerous cells [17].

Human papillomaviruses exhibit resistance to chemical

Corresponding Author:
Dr. Manoochehr Makvandi
Associate Professor, Department of Restorative Dentistry, School of Dentistry, Ahvaz Jundishapur University of Medical Sciences, Ahvaz, Iran.
Email: manoochehrmakvandi299@gmail.com

disinfectants, such as glutaraldehyde solution and ortho-phthalaldehyde, and are only effectively inactivated by agents such as, hypochlorite, and PAA-silver-based disinfectants [18, 19].

Each year, high-risk (HR) human papillomavirus (HPV) groups account for approximately 630,000 novel oncological cases, with cervical cancer representing the majority at 530,000 instances, which equates to 4.5% of all human malignancies globally [20]. Furthermore, Low risk human papillomavirus (LR-HPV) serves as the causative agent for over thirty million cases of genital warts [18]. The predominant mechanism of mucosal HPV transmission occurs through intimate sexual interactions; nevertheless, non-sexual transmission of HPV may transpire through contaminated instruments such as colposcopes, colonoscopy devices, transvaginal ultrasound probes, endoscopes, and speculums [18]. Specifically, the contamination of fomites predominantly occurs during the course of clinical examinations. Consequently, the establishment of effective disinfection protocols is essential for the reduction of inadvertent HPV transmission.

There currently exists treatment for LR-HPV. The prevailing therapeutic approaches encompass podophyllotoxin [21], Imiquimod [22], Sinecatechins [23], intralesional/topical interferon [24], and topical 5-aminolevulinic acid (ALA) [25] for the management of genital and anal warts. The HPV vaccination initiative is effective solely against low-risk (LR) HPV and high-risk (HR) HPV types. The current treatment for HR-HPV is: with neoadjuvant cisplatin-based chemotherapy with high recurrent rate of 25-40% [26] and Multiple monoclonal antibodies (mAb) such as Ipilimumab, Pembrolizumab and Nivolumab for targeting of the PD-1 axis in cervical cancer [27].

Human Papillomaviruses (HPVs) are classified as DNA viruses that predominantly infect human hosts and are associated with various malignancies, most notably cervical carcinoma in females. The documentation of HPV infection has elucidated the involvement of HPVs in the pathogenesis of head and neck squamous cell carcinoma, esophageal carcinoma [28], cervical carcinoma [29], head and neck malignancies [30], as well as brain [31] and pulmonary neoplasms [32]. The identification of independent traditional risk factors, diverse clinical manifestations, and heightened prevalence among specific demographic groups and geographical locales has engendered a growing interest in the epidemiology of HPV infection.

The promotion of awareness and implementation of vaccination programs against HPV are of paramount importance, as a significant proportion of young adults remain uninformed regarding the associated risks and advantages of vaccination. The preventative HPV vaccines were formulated utilizing L1 protein derived from recombinant DNA technology. The immune response elicited by the vaccine was characterized by the production of neutralizing immunoglobulin G antibodies and the activation of cellular immunity [33]. The commercially available vaccines include Cervarix™ and Gardasil®. These two vaccines differ in their formulations. The Gardasil® vaccine specifically targets HPV types including type 6, type 11, type 16, and type 18, and was approved in Europe and US [34]. The Gardasil®9 vaccine is designed to address five additional HPV strains (HPA31, HPA33, HPA45, HPA52, and HPA58) beyond the scope of the original Gardasil® vaccine. The introduction of a bivalent vaccine, which targets HPV 16 and HPV 18, occurred in both the United States and Europe in 2009 [35]. Administration of the 9vHPV vaccine has been shown to potentially avert 3% of laryngeal cancer cases, 4% of oral cavity cancers, 21% of oropharyngeal cancers, 23% of vulvar cancers, 25% of penile cancers, 61% of vaginal cancers, 79% of anal cancers, and 90% of cervical cancers [36]. As of June 2020, over 55% of World Health Organization member states had commenced HPV vaccination initiatives through national immunization programs. A growing body of research conducted across 22 nations has examined the effectiveness of the 9vHPV and 4vHPV vaccines in reducing the prevalence of HPV infections and associated disease in human populations [35, 37].

Herpes family

The family of human herpesviruses is characterized by enveloped double-stranded DNA genomes ranging from 125,000 to 240,000 base pairs in length. To date, only nine subtypes have been identified as having the capacity to infect humans (9). These include Herpes Simplex Viruses types 1 and 2 (HSV-1 and HSV-2), Varicella-Zoster Virus (HHV-3), Epstein-Barr Virus (EBV or HHV-4), Human Cytomegalovirus (HCMV or HHV-5), Human Herpesvirus types 6A and 6B (HHV-6A and HHV-6B), Human Herpesvirus-7 (HHV-7), and Kaposi's Sarcoma-Associated Herpesvirus (HHV-8) [38].

These viruses cause disease, cold sores [39], genital herpes, stromal keratitis, malignancies [40], meningitis, and encephalitis [41, 42]. All herpesviruses undergo two distinct replication cycles: lytic and latent [42]. The lytic replication cycle culminates in the generation of viral particles that propagate infection to other cells and organisms, whereas the latent phase is characterized by restricted gene expression and the absence of infectious particles. Herpesviruses establish latency in various anatomical sites and have the potential to elicit disease during both the initial infection and subsequent reactivation; however, the underlying mechanisms that prompt latency and reactivation, remain inadequately understood [38].

Herpes viruses' transmission via saliva

Herpes viruses included, Herpes simplex virus type 1 (HSV-1) [43], Herpes simplex virus type 2 (HSV-2) [44], Epstein Barr virus [44], Human cytomegalovirus [45], Human Herpesvirus types 6 [46] and Human Herpesvirus types 8 [47] are transmitted via saliva of infected individuals during dental practice.

Herpes simplex virus type 1 and Herpes simplex virus type 2

Both HSV-1 and HSV-2 are highly transmissible via saliva that propagate readily during active viral shedding [43, 44]. The presence of moist vesicles on the mucosal surfaces of the lips, oral cavity, and genital regions signifies an active infection. Both viruses undergo two distinct replication cycles: lytic and latent. during lytic cycle cause oral herpes or Cold sores, meningitis and encephalitis [41, 42]. Infections caused by HSV-1 and HSV-2 do not resolve spontaneously; rather, they persist as lifelong latent infections that may precipitate recurrent episodes [48]. The administration of antiviral medications can ameliorate the severity of reactivation episodes and diminish the likelihood of transmission [49]. HSV-1 was detected in dental clinic [50]. Although oral HSV-1 and HSV-2 infections are recognized within the context of dental practice, they garner comparatively less clinical attention due to their lower incidence among patients and the diagnostic complexities involved. At present no vaccine available against HSV-1 and HSV-2.

Varicella Zoster

Varicella-zoster virus (VZV) represents a human alphaherpesvirus that is responsible for the manifestation of chickenpox (varicella) and shingles (herpes zoster) [75]. VZV possesses a linear, double-stranded DNA genome spanning approximately 125 kilobases, which encodes a minimum of 71 distinct proteins [51].

The transmission of chickenpox occurs via direct contact with the blisters, saliva, or mucus of an individual infected with the virus. Additionally, the virus may be disseminated through airborne particles resulting from coughing and sneezing. VZV has the potential to induce ulcerations within the oral cavity [38].

Varicella zoster virus (VZV) initiates varicella (commonly referred to as chickenpox) as the primary infection, while zoster (known as shingles) arises from the reactivation of the virus following latency within sensory ganglion neurons [52]. The complications associated with Varicella zoster virus encompass bacterial superinfection of the integumentary system, encephalitis, and pneumonia. Patients who present with zoster should receive prompt treatment with antiviral agents such as acyclovir, famciclovir, or valacyclovir, which are administered via the oral route [52]. VZV has been identified in a dental clinic setting [53]. Varicella vaccine available for children and adult [54].

Epstein Barr Virus

Epstein–Barr virus (EBV), commonly identified as human gammaherpesvirus 4, represents a double-stranded DNA (dsDNA) virus categorized within the Herpesviridae family. The ubiquity of EBV within global populations can be attributed to its various transmission mechanisms and its capacity to integrate into the genomic structure of B cells, thereby facilitating persistent infections [55].

Transmission of EBV occurs predominantly through saliva, with the virus initially targeting the epithelial cells located in the oropharynx, nasopharynx, and tonsillar regions for its transcriptional and replicative processes, subsequently proceeding to infect B lymphocytes via specific envelope glycoproteins [56]. Following the initial infection, EBV transitions into a latent state, residing in resting memory B cells, where the replication of EBV is regulated by the host's immune response. A decline in immune function can precipitate the reactivation of EBV, leading to a renewed lytic phase and the generation of infectious virions. This sequence of events prompts a new cycle of epithelial infection in the tonsils, accompanied by viral shedding in the saliva [57].

Approximately 35-50% of the adolescent demographic within the human population experiences the onset of infectious mononucleosis (IM), after which the virus remains latent throughout the individual's lifespan [58]. Nevertheless, when the equilibrium between the virus and the host's immune response is perturbed, Epstein-Barr virus (EBV) can instigate malignant transformation in both lymphoid and epithelial cells, resulting in approximately 200,000 fatalities each year [59].

The Epstein-Barr virus asymptomatically infects over 95% of healthy adults globally [60]. The Epstein-Barr virus (EBV) has been associated with the pathogenesis of a diverse array of human malignancies, which include Burkitt lymphoma, nasopharyngeal carcinoma, T/NK lymphoma, gastric cancers, B cell lymphoma, Hodgkin's disease, certain T cell lymphomas, post-transplant lymphoproliferative disease, and, more recently, specific cancers affecting the stomach and smooth muscle [60, 61]. EBV was detected in patients with periodontal diseases in dental clinic [62]. Currently, there exists no targeted therapeutic intervention against EBV. Furthermore, there is neither an approved nor a therapeutic vaccine available for the Epstein-Barr virus.

Human cytomegalovirus

Human cytomegalovirus belongs to Herpesviridae, or alternatively referred to as human herpesvirus-5 (HHV-5) [63]. Cytomegalovirus (CMV) is classified as a double-stranded DNA virus and is categorized within the herpesvirus family. This ubiquitous virus exhibits a spectrum of clinical manifestations, ranging from asymptomatic presentations to severe end-organ dysfunction, particularly in immunocompromised individuals suffering from congenital CMV disease [64]. Congenital cytomegalovirus infection has the capacity to induce significant morbidity and may even result in mortality [65].

While CMV infection may remain asymptomatic in immunocompetent individuals, it possesses the potential to be life-threatening for patients with compromised immune systems [65]. The saliva and urine of young children are major sources of virus [66]. In industrialized nations, CMV infects approximately 45% of the adult population, whereas in emerging countries, the infection rate approaches nearly 100% [67]. The presence of CMV constitutes a lifelong challenge characterized by antigenic T-cell surveillance and accompanying immune dysfunction [68]. Diseases causes by CMV in immunocompromised

patients are, hepatitis, Graft rejection, vascular diseases, hepatitis, gastrointestinal ulceration, retinitis pneumoniae and autoimmune diseases [69, 70]. Subsequent to initial infection, CMV typically persists in a latent state; however, it is capable of reactivation at any point in time [69,70]. Ultimately, it has been implicated as a possible contributing factor in the development of breast, colorectal muscle, brain, and prostate cancers [71, 72]. Human cytomegalovirus was detected among patients in dental clinic [73]. CMV-specific hyperimmunoglobulin (CMVIG) as off-label therapy, valacyclovir, ganciclovir is used for CMV treatment [74]. Currently, no approved vaccine available to prevent Congenital cytomegalovirus infection.

Human herpesvirus 6 (HHV-6)

Human herpesvirus 6 is categorized into two distinct entities, namely Human herpesviruses 6A and 6B (HHV-6A/6B; species Human betaherpesvirus 6A and Human betaherpesvirus 6B). HHV-6A/6B is a member of the Herpesviridae family [75]. Transmission of HHV-6A/6B occurs primarily through salivary secretions [76]. The mature CD4+ T cell serves as the primary target for HHV-6 infection. The virus exhibits pleiotropic influences on the immune system, notably including the modulation of natural killer cell functionality [77]. The occurrence of HHV-6A is notably more prevalent in individuals with compromised immune systems. Conversely, investigations have identified HHV-6B as the causative agent of the pediatric condition known as exanthema subitem (roseola infantum) [77]. HHV-6 is implicated in the etiology of acute febrile illnesses [77], hepatitis [78], encephalitis [79], and graft rejection phenomena [80]. Moreover, HHV-6 has been identified in the saliva of patients attending dental clinics [81].

In the context of patients undergoing stem-cell transplantation, the administration of Ganciclovir has shown significant therapeutic advantages and is regarded as the antiviral treatment of choice [82]. At present, there exists no FDA-approved therapeutic agent specifically designated for the treatment of HHV-6, nor is there a commercially available vaccine [83].

Influenza A and B virus

Influenza is classified by the World Health Organization (WHO) as a "serious global health threat" and accounts for a considerable number of fatalities on a global scale [84]. The Influenza A virus (IAV) and Influenza B virus, which are the primary etiological agents responsible for influenza, are classified within the Orthomyxoviridae family, characterized by segmented, negative-sense, single-stranded RNA viruses [85]. Among the various influenza viruses, the influenza A viruses (IAVs) are the most virulent and significant, being responsible for seasonal epidemics and all prior influenza pandemics [86]. The Influenza A virus is implicated in lower respiratory tract infections; it also possesses the broadest host range, highest morbidity and mortality rates, and is capable of inducing the most severe clinical manifestations [85]. The transmission of Influenza viruses occurs through saliva [87]. The presence of the Influenza virus has been identified in dental clinics [88]. The administration of Oseltamivir is associated with improved survival rates in hospitalized patients suffering from pneumonia caused by human influenza A/H3N2, A/H1N1, or B viruses [89]. Since dental students and dentists at risk of acquiring influenza infection, influenza vaccination program is a vital and shows very effectiveness against human influenza A/H3N2, A/H1N1, or B viruses. Dent team should receive a single dose of flu vaccine every year [90].

Covid 19

On March 11, 2020, the World Health Organization (WHO) formally declared the emergence of a pandemic associated with the novel coronavirus, Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), presently referred to as COVID-19 [91]. The COVID-19 pandemic has precipitated significant global ramifications, impacting hundreds of millions of individuals and resulting in over six million fatalities [92].

Variants of the SARS-CoV-2 virus, namely Alpha, Beta, Delta, and the extensively mutated Omicron lineage, have instigated successive waves of infections following the global dissemination of the original strain since the year 2020 [93]. Omicron variants such as BA.2.86 and JN.1 exhibit in excess of 100 mutations that confer immune evasion capabilities. Specifically, Omicron BA.2.86 displays resistance to class 2 and 3 monoclonal antibodies targeting the receptor-binding domain, while also demonstrating a heightened receptor affinity in comparison to XBB.1.5 or EG.5.1. The novel subvariant BA.2.86, designated JN.1, manifests over 100 mutations relative to the wild type, indicative of a potential evolutionary divergence [94]. The transmission of SARS-CoV-2 occurs predominantly through the saliva of infected individuals [95]. Given that there exists no definitive therapeutic intervention for SARS-CoV-2 (COVID-19) [96], it follows that the COVID-19 vaccination initiative, encompassing mRNA vaccines, represents the sole effective strategy identified to mitigate the impact of SARS-CoV-2 [96, 97].

*Hepatitis B, hepatitis C viruses and human immunodeficiency virus (H*IV)

An essential mode of transmission within the dental practice is the direct contact with blood and bodily fluids [98]. Gingival bleeding often results in the presence of blood within saliva; consequently, it is imperative that all saliva be regarded as potentially infectious material [98]. Notable bloodborne pathogens of significance encompass the hepatitis B and C viruses, as well as the human immunodeficiency virus. The transmission of HBV within the field of dentistry occurs predominantly through percutaneous injury, which is associated with an average risk of HBV transmission estimated at 30%. The Hepatitis B virus is capable of retaining its infectious nature in desiccated blood at ambient temperature for a duration of one week [98].

Hepatitis B Virus (HBV)

The Hepatitis B virus is classified as an enveloped DNA virus and is a member of the Hepadnaviridae family [99]. An estimated 296 million individuals are currently afflicted with chronic Hepatitis B virus (HBV) infection on a global scale. Moreover, annual mortality attributed to liver cirrhosis and hepatocellular carcinoma (HCC) reaches approximately 820,000 individuals [99]. The transmission of HBV is facilitated through percutaneous injury, which is associated with an average risk of transmission amounting to 30% [98]. The presence of HBV among dental professionals has been documented in Iran [100]. Currently, entecavir (ETV), tenofovir disoproxil fumarate (TDF), tenofovir alafenamide (TAF), and pegylated interferon (PEG-IFN) are preferred initial treatment for anti-HBV therapy [99]. The effective hepatitis B vaccine is available against hepatitis B virus which has declined significantly the rate of chronic HBV globally [101].

Postexposure management for hepatitis B virus

Post-exposure management for the hepatitis B virus (HBV) necessitates the administration of multiple doses of hepatitis B immune globulin (HBIG) in conjunction with the hepatitis B vaccine [101]. HBIG is a preparation of human immunoglobulin utilized to inhibit the onset of hepatitis B subsequent to acute exposure to the surface antigen of the hepatitis B virus (HBsAg) [102]. The antibody levels of the individual exposed should be evaluated if the source exhibits a positive HBsAg status. In instances where the exposed individual demonstrates a lack of immunity (i.e., has not been vaccinated, failed to achieve seroconversion post-vaccination, or has antibody titers to HBsAg below 10 mIU/mL), the recommended intervention is the administration of a single dose of HBIG within a timeframe of 48 to 72 hours post-exposure, alongside the initiation of the HBV vaccination series [101]. The HBV vaccine should be administered within a span of seven days following exposure, with subsequent doses given one or two months later, and then six months after the initial dose. The level of immunity should be assessed two to four weeks thereafter [101].

The risk of transmission of hepatitis B virus (HBV) exceeds 30% in situations where the source is positive and post-exposure prophylaxis is not administered [101, 102].

Hepatitis C viruses

The Hepatitis C virus (HCV) is categorized as an enveloped positive-sense RNA virus and belongs to the Flaviviridae family [103]. HCV impacts over 170 million individuals worldwide and serves as a common etiological contributor to acute hepatitis, chronic hepatitis, and hepatocellular carcinoma [104]. Approximately 400,000 individuals worldwide perish as a result of HCV-related conditions each year [105]. The transmission of HCV primarily occurs via percutaneous injuries, such as needle sticks or exposure to blood, tissue, and other bodily fluids, which presents an average risk of HCV transmission estimated at 0.2% [106]. Evidence of HCV infection has been documented among dental professionals [107]. The emergence of safe and effective oral direct-acting antivirals (DAAs) for the treatment of hepatitis C holds significant promise for the potential eradication of Hepatitis C Virus [108]. Currently, there exists no approved vaccine for the prevention of Hepatitis C Virus infection [109].

Postexposure management for hepatitis C Virus

Following exposure to the hepatitis C virus, it is imperative that the affected healthcare professional undergoes meticulous follow-up and is referred for suitable therapeutic interventions should infection be confirmed. Currently, there exists no efficacious post-exposure prophylaxis (PEP) for hepatitis C [101].

The probability of transmission subsequent to an injury inflicted by a positive source is contingent upon whether active viral replication is present. In instances where the source is negative for HCV RNA as determined by polymerase chain reaction (PCR) testing, the transmission risk ranges from 1.8% to 3.1%. Conversely, if the source is PCR positive, the transmission risk escalates to 10%. It is advisable to conduct testing at baseline, at three months, and at six months post-exposure. Additionally, liver function assessments, including aminotransferase (ALT) and aspartate aminotransferase (AST), are recommended at two, three, and six months. Continuous monitoring of clinical manifestations should be performed by an infectious disease specialist or gastroenterologist [101].

In June 2016, the World Health Organization (WHO) released its inaugural global health sector strategy, articulating the objective of eradicating viral hepatitis B and C as a significant public health concern by the year 2030 [110].

HIV

The human immunodeficiency virus (HIV) is an enveloped virus that consists of two positive single-stranded RNA molecules and is classified within the Retroviridae family [111]. To date, two distinct types of HIV, namely HIV-1 and HIV-2, have been identified [112]. The human immunodeficiency virus (HIV) constitutes a significant global public health challenge [111]. It was estimated that in the year 2019, approximately 38 million individuals were living with HIV, with an additional 1.7 million individuals newly diagnosed [113]. In the Islamic Republic of Iran, akin to numerous other nations, the incidence of HIV infection has escalated swiftly in recent years [114, 115]. Reports from the Iranian Ministry of Health and Medical Education indicated that 22,406 HIV-positive individuals resided in Iran in the year 2019 [116]. The primary mode of HIV transmission is through sexual contact, by maternal-infant exposure, percutaneous injuries [117]. At present, no definitive therapeutic interventions exist for HIV-1 and HIV-2 [117]. Furthermore, there is currently no licensed vaccine available for the prophylaxis of HIV infection [118].

Postexposure management for HIV

The probability of seroconversion following a sharps

injury involving HIV-contaminated blood is estimated at 0.3%, whereas the risk associated with exposure of mucous membranes to HIV-infected blood is quantified at 0.09% [119]. Consequently, it is imperative that post-exposure testing and medical evaluation are conducted.

In instances where the source may be infected with HIV, the consideration of post-exposure prophylaxis (PEP) is warranted. The primary objective of PEP is to inhibit viral replication. The fundamental regimen for HIV PEP typically incorporates zidovudine (ZDV) in conjunction with lamivudine (3TC). Alternatively, the regimen may consist of 3TC paired with stavudine, or didanosine combined with stavudine as appropriate. The enhanced regimen consists of the primary regimen supplemented with indinavir, nelfinavir, efavirenz, or abacavir [120].

The potential toxicity and adverse effects associated with HIV PEP warrant careful consideration. PEP is indicated solely in cases of substantial exposure, accompanied by a thorough risk assessment. Administration of the pharmacological agents should ideally occur within a timeframe of 24 to 36 hours following exposure [119].

Postexposure testing for HIV antibodies is recommended at baseline, six weeks, 12 weeks, and six months postexposure [120]. Follow-up includes reporting any sudden or severe flu-like symptoms, any adverse events associated with PEP if this is administered, as well as signs and symptoms of possible retroviral illness.

If the status of the source is unknown, decisions should be based on the exposure risk and the chance that the source may be HIV positive, e.g. an IV drug user. If the source consents to HIV testing, post-exposure prophylaxis can be given as soon as possible after exposure and discontinued if the result is negative [120].

Enhancing dental team outcomes

Due to the inherent characteristics of their profession, dental practitioners encounter potential infectious agents on a daily basis, which constitutes a significant occupational hazard. It is imperative that vaccination protocols, appropriate utilization of personal protective equipment (PPE), as well as disinfection and sterilization guidelines, be meticulously adhered to in order to mitigate the transmission of infections [121]. Comprehensive protocols designed to reduce percutaneous injuries must be established within the dental practice, thoroughly elucidated to newly inducted personnel, and subject to regular review and assessment. In the event of an incident, a clearly defined exposure protocol and risk assessment should be implemented [101]. Infection control methodologies, ongoing educational initiatives, and vaccination efforts must be consistently emphasized, as both dental healthcare providers and their patients face the risk of infections and the potential for disease transmission. Effective sterilization techniques are essential to avert cross-contamination between patients [101, 121].

The critical significance of infection control must remain at the forefront of our considerations: during clinical interactions, while treating each patient, when transitioning between clinical activities, while processing dental instruments, and upon exiting the office toward home. This will minimize the risk of pathogens transmission and ensure patient and personnel safety.

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- Author declares no conflict of interest
- Study was approved by Research Ethic Committee of author affiliated Institute.
- Study's data is available upon a reasonable request.
- All authors have contributed to implementation of this research.

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