STM JOURNALS

Recent Trends in
Infectious Diseases

ISSN: 3048-9857
Volume 2, Issue 1, 2025
January—June
DOI (Journals): 10.37591/RTID

https://journals.stmjournals.com/rtid

Review RTID

A Comprehensive Review on Monkey Pox Infection

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Abstract

Monkeypox represents a zoonotic viral affliction instigated by the monkeypox virus, which is classified within the Poxviridae family and is distinct from the virus that causes chickenpox. Initially recognized in 1958, this infection has recently attracted international scrutiny due to its escalating incidence beyond traditional endemic territories. This review delivers an exhaustive examination of monkeypox, encompassing its epidemiology, viral morphology, modes of transmission, pathogenesis, clinical presentations, and therapeutic interventions. The virus, which exhibits structural similarities to smallpox, is characterized as a substantial, enveloped, double-stranded DNA virus. Transmission occurs via proximate interaction with infected animals or individuals, as well as through contaminated materials. Pathogenesis entails the viral ingress through mucosal surfaces, followed by replication and systemic dissemination. Clinical manifestations typically encompass fever, rashes, and lymphadenopathy, in contrast to the typical presentation of chickenpox. Diagnosis predominantly hinges upon polymerase chain reaction (PCR) testing, with emerging techniques such as loop-mediated isothermal amplification (LAMP) and recombinase polymerase amplification (RPA) also demonstrating the potential for the detection of the monkeypox virus. These newer diagnostic methods, like RPA, offer faster and more accessible alternatives to traditional PCR testing. Therapeutic alternatives include antiviral agents such as Cidofovir, Brincidofovir, and Tecovirimat, which have demonstrated effectiveness against orthopox viruses. This review underscores the necessity for augmented surveillance, enhanced community awareness, and ongoing research to confront the challenges presented by monkeypox. It accentuates the significance of international collaboration in curbing the dissemination of the virus and safeguarding public health.

Keywords: Monkeypox, zoonosis, orthopox virus, epidemiology, antiviral treatment

INTRODUCTION

Monkeypox constitutes a zoonotic viral affliction instigated by the monkeypox virus, which manifests as a dermatological eruption analogous to that of smallpox. The initial documentation of monkeypox occurred in 1958 amidst the transport of monkeys from Singapore to Denmark (Figure 1) [1]. Nevertheless, the inaugural confirmed instance of human infection transpired in 1970, when the virus was successfully isolated from a child in the Democratic Republic of Congo, who was suspected to be afflicted with smallpox [2]. The monkeypox virus is classified within the Poxviridae family and the Chordopoxvirinae subfamily [3]. The designation "monkey virus" is derived from its original identification in monkeys [4]. A variety of rodent species indigenous to the tropical rainforests of Central and West Africa, including tree squirrels and Gambian pouched rats, are presently acknowledged as reservoirs for the monkeypox virus. African apes and monkeys possess susceptibility to infection and are posited to act as intermediate hosts [5]. Two distinct genetic clades of the monkeypox virus have been identified: Clade I and Clade IIa. The strain responsible for the current outbreak is categorized as Clade IIb [6]. The

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Received Date: September 14, 2024
Accepted Date: September 25, 2024
Published Date: October 01, 2024

Citation: Sinchana Bhat, Ramdas Bhat. A Comprehensive Review on Monkey Pox Infection. Recent Trends in Infectious Diseases. 2025; 2(1): 23–32p.

transmission of the monkeypox virus (MPV) occurs through contact with the respiratory droplets, skin lesions, or bodily fluids of infected animals. Reports indicate that patients initially exhibit signs or symptoms, including rash (97%), fever (85%), adenopathy (71%), and chills (71%), which collectively characterize the preliminary clinical presentation of monkeypox viral infection [7]. The incidence and geographical prevalence of human monkeypox cases have escalated in recent years, thus being acknowledged as a burgeoning public health concern, particularly in regions of West Africa where there exists substantial interaction between humans and wild animal reservoirs, and specifically where there is substantiated evidence of an escalating infection attack rate [6]. This review endeavors to provide an exhaustive examination of monkeypox virus infection, encompassing its epidemiology, structural characteristics, transmission mechanisms, pathogenesis, clinical manifestations, diagnostic approaches, and the therapeutic agents employed in the treatment of the disease.

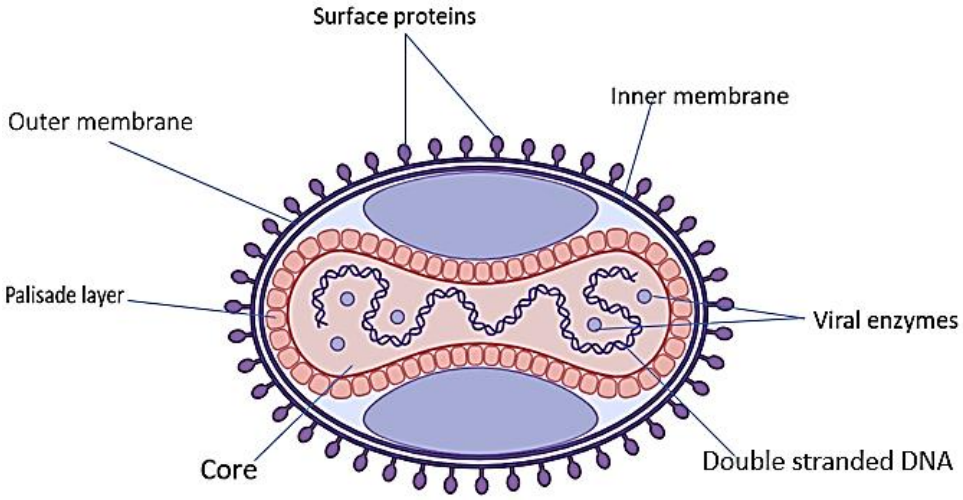

Figure 1. Structure of Monkeypox virus.

EPIDEMIOLOGY

The Monkeypox outbreak of 2022 is characterized by numerous atypical features regarding the incidence of positive cases. As of September 4, 2023, there have been 89,752 verified instances of Monkeypox virus infection, culminating in 157 fatalities across 113 nations, thereby establishing it as a notable concern due to its high transmissibility and association with various human health issues. Based on data from the World Health Organization (WHO), the demographic most affected comprises individuals aged 27 to 41, accounting for 79.1% of cases; a substantial proportion of the affected population is male, representing 96.3%, and 52.5% of those infected are HIV-positive [8]. Annual statistics about Monkeypox cases and mortality from 2018 to 2021 were meticulously gathered from national surveillance systems, WHO bulletins, and published case reports or outbreak analyses, subsequently validated in collaboration with national surveillance teams; these figures are presented alongside human Monkeypox case report data dating back to 1970. Since the year 2018, cases have been documented in six African nations: Cameroon, the Central African Republic (CAR), the Democratic Republic of the Congo (DRC), Nigeria, the Republic of the Congo (ROC), and Sierra Leone, with the DRC reporting over 3,000 suspected cases annually and reaching a zenith of 6,216 cases and 222 deaths in 2020 [9]. Since January 1, 2022, 110 Member States have communicated instances of Monkeypox to the WHO across all six WHO regions. A cumulative total of 82,768 laboratory confirmed Monkeypox cases and 65 deaths were recorded by the WHO from 110 countries throughout all six WHO regions between January 1, 2022, and December 15, 2022 [10]. But as of 2023, the monkeypox outbreak has seen a decline in cases compared to the peak in 2022. In 2024, the monkeypox situation generally stabilized, with significantly fewer cases reported compared to the previous outbreaks. Continued vaccination efforts and public health campaigns have contributed to this decline [11].

STRUCTURE OF VIRUS

The Monkeypox virus, which is categorized as a large and morphologically distinctive pathogen, exhibits a brick-shaped or ovoid appearance and is characterized by its enveloped structure, possessing a double-stranded linear DNA (dsDNA) genome that is approximately 190 kilobases in length and features a diameter that ranges between 200 and 250 nanometers, as indicated in the referenced literature [12]. Furthermore, this virus is comprised of a core region that contains lateral bodies along with a surrounding lipoprotein envelope, which plays a critical role in its structural integrity and function [13]. Viral architecture is characterized by the presence of two distinct membranes; specifically, an inner membrane and an outer membrane, each of which is associated with unique proteins, namely, the inner membrane proteins and surface proteins, respectively, that contribute to its pathogenicity and interaction with host cells. Notably, the Monkeypox virus exhibits a unique replication mechanism, wherein it specifically undergoes its replication process within the cytoplasmic compartment of the host cells that it infects, as opposed to the more common nuclear replication seen in other viral pathogens. During the process of viral replication within the infected cells, two morphologically and functionally distinct forms of viral particles are generated, namely, the intracellular mature virus (IMV) and the extracellular enveloped virus (EEV), each exhibiting unique structural characteristics and modes of transmission. The IMV form is characterized by the presence of a single inner membrane, while the EEV variant possesses two membranes that are derived from the endoplasmic reticulum, thus highlighting their differences in structural composition. Both viral forms are capable of initiating infection in susceptible host cells, although they utilize divergent mechanisms to facilitate this infective process. Additionally, the genetic material of the virus is complex and encompasses more than 200 open reading frames (ORFs), which encode various proteins essential for its replication and pathogenesis, as noted in the cited references [12].

TRANSMISSION

The transmission dynamics of monkeypox encompass not only the transfer of the virus from animal hosts to human beings but also the subsequent human-to-human transmission pathways, highlighting the complex interplay of epidemiological factors involved in the spread of this disease (Figure 2).

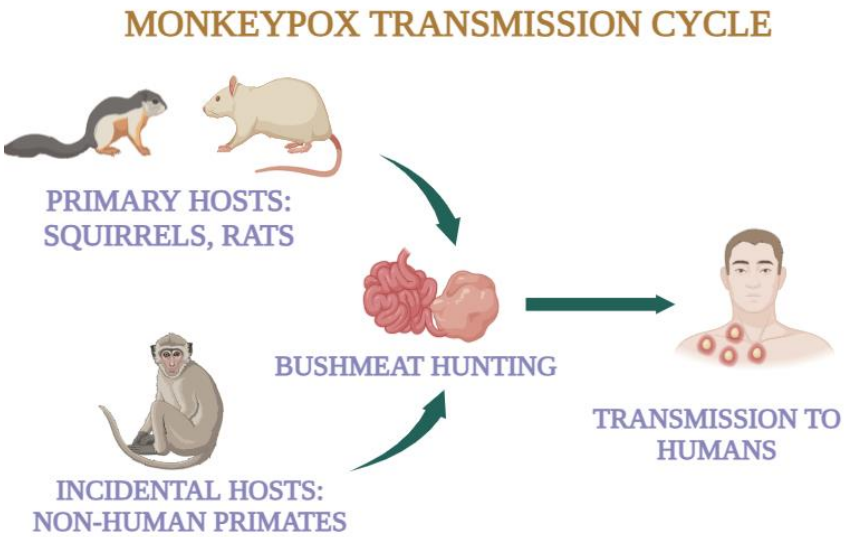

Figure 2. Monkeypox transmission cycle.

Humans may become susceptible to infection through a variety of avenues, which include, but are not limited to, direct contact with animals that have been infected with the virus, such as instances where individuals engage in the handling or consumption of meat derived from these animals, or through interactions with materials and surfaces that have been contaminated by the virus. Proximity to infected animals, along with exposure to their bodily fluids, including blood, saliva, or urine – constitutes a

significant mechanism by which monkeypox is transmitted from animals to humans, thereby underscoring the critical importance of understanding these vectors in the context of public health [14]. Furthermore, individuals who engage in sexual relationships with multiple partners are identified as being at an elevated risk for the acquisition of pox, suggesting that behavioral factors play a crucial role in the transmission of this virus among populations. Additionally, the transmission of monkeypox can occur through contact with contaminated items, which may include clothing or linen that has been soiled, as well as through needle stick injuries that take place within healthcare settings, or in communal environments such as tattoo parlors where hygiene practices may be insufficient. It is also noteworthy that the virus has the potential to be transmitted to a fetus during pregnancy or at the time of birth, indicating that vertical transmission is a relevant consideration in the epidemiology of monkeypox. The process of animal-to-human transmission of monkeypox is facilitated by direct interactions with infected animals, which may occur through bites or scratches inflicted by these animals, or during various activities that involve the handling of these animals, such as hunting, skinning, trapping, cooking, playing with carcasses, or consuming the flesh of these infected animals [15]. Moreover, the likelihood of infection increases through engagement in activities that augment exposure to animals, such as sleeping outdoors on the ground or frequenting areas near forested regions, which may serve as habitats for the reservoirs of the virus [4]. Recent investigations have also yielded the detection of the monkeypox virus in semen samples, as confirmed by researchers, indicating that sexual transmission may be a potential route for the spread of this pathogen [16].

PATHOGENESIS

Initial Infection

The monkeypox virus typically enters the human body through close contact with infected animals or humans. The virus gains entry via mucous membranes, particularly those of the respiratory tract or oropharynx. In some cases, it may also enter through small breaks in the skin (Figure 3).

Primary Replication

Once the virus enters the body, it begins to be replicated at the site of inoculation. This initial replication occurs in the epithelial cells of the respiratory tract and oropharyngeal mucosa. During this stage, the host may not show any symptoms, as the virus is establishing its presence and multiplying.

Spread to Lymph Nodes (Primary Viremia)

As the virus replicates, it spreads to nearby lymphoid tissue, particularly the regional lymph nodes. This spread, known as primary viremia, allows the virus to establish a foothold in the lymphatic system. The virus continues to replicate in these lymphoid tissues, increasing its numbers significantly.

Systemic Spread (Secondary Viremia)

After multiplying in the lymph nodes, the virus enters the bloodstream in larger numbers, leading to secondary viremia. This stage allows the virus to spread throughout the body, infecting various organs and tissues, including the skin, liver, spleen, and other lymphoid tissues.

Incubation Period

The time between the initial infection and the appearance of symptoms is called the incubation period. For monkeypox, this typically lasts 7 to 14 days but can extend up to 21 days in some cases. During this time, the virus is actively replicating and spreading, but the host's immune system has not yet mounted a significant response.

Cellular Entry and Replication

At the cellular level, monkeypox virus, like other pox viruses, has two main methods of entry:

a. *Low pH endosomal pathway:* The virus is engulfed by the cell and enters via endosomes, with the acidic environment triggering viral membrane fusion.
b. *Direct fusion:* The virus can fuse directly with the cell membrane at neutral pH.

Once inside the cell, the virus releases its core into the cytoplasm. This core contains viral DNA and essential enzymes for replication.

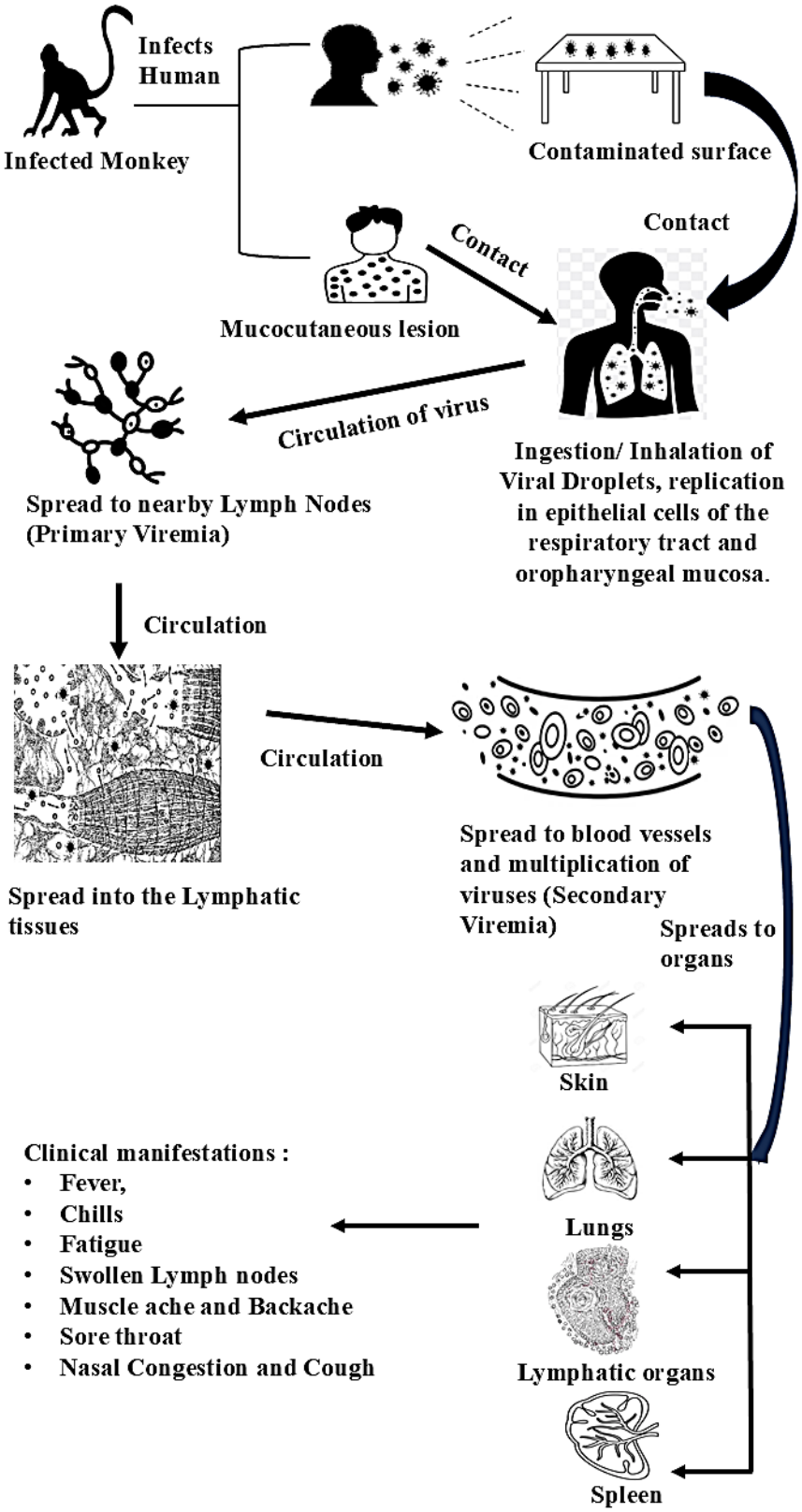

Figure 3. Pathogenesis of Monkeypox viral infection.

Viral Gene Expression and Protein Synthesis

The virus begins its replication cycle by expressing its genes in a carefully orchestrated sequence:

a. *Early gene expression:* Initiated by a viral DNA-dependent RNA polymerase, which is packaged within the viral particle.
b. *Intermediate gene expression:* Follows early gene expression and requires newly synthesized viral proteins.
c. *Late gene expression:* Occurs after DNA replication has begun and produces structural proteins for new viral particles.

These viral mRNAs are translated on host ribosomes, producing the proteins necessary for viral replication and assembly.

DNA Replication

Viral DNA synthesis occurs in specialized cytoplasmic structures called "factories". These start as compact DNA-containing structures surrounded by endoplasmic reticulum membranes. As replication progresses, they transform into crescent-shaped structures where new virions are assembled.

Virion Assembly and Maturation

New viral particles are assembled in these cytoplasmic factories. Most of these mature virions remain inside the cell as intracellular mature virions (IMVs). However, some undergo additional processing.

a. Some IMVs are transported along microtubules and acquire two additional membrane layers from the endoplasmic reticulum or Golgi apparatus, becoming intracellular enveloped virions (IEVs).
b. IEVs can then move to the cell surface using viral proteins that induce actin polymerization, forming actin tails that propel the virions towards adjacent cells.
c. When IEVs reach the cell surface, their outermost membrane fuses with the plasma membrane, releasing cell-associated enveloped virions (CEVs) or extracellular enveloped virions (EEVs) [17].

SIGNS AND SYMPTOMS

Rashes can appear on the hands, feet, chest, face, mouth, or near the genitalia in those who have monkeypox. Before healing, the rashes will go through a few stages, including scabs. The rashes may be unpleasant or itchy at first, and they may resemble pimples or blisters [18]. Around distinct lesions, erythematous or hyperpigmented areas of skin might be observed. Detached scabs may be much smaller than the original lesion. It is also possible to observe inflammation of the vaginal, conjunctival, and pharyngeal mucosae [6].

Other symptoms include [18]:

- Fever.
- Chills.
- Swollen lymph nodes.
- Exhaustion.
- Muscle aches and backache.
- Headache.
- Respiratory symptoms (e.g., sore throat, nasal congestion, or cough).

DIAGNOSIS

Polymerase Chain Reaction (PCR) is a useful tool for detecting the monkeypox virus [19]. The World Health Organization (WHO) recommends this test for confirmation of monkeypox virus infection because of its excellent sensitivity and specificity, making it the gold standard in the field. In addition, the identification of monkeypox infection is also accomplished using Nucleic Acid Amplification Tests (NAATs) such as Recombinase Polymerase Amplification Assay (RPA) and Loop-mediated isothermal amplification (LAMP) [20].

Polymerase Chain Reaction (PCR)

The polymerase chain reaction, commonly abbreviated as PCR, represents an advanced molecular biology technique that facilitates the exponential amplification of specific nucleic acid sequences derived from a designated target, which significantly enhances the ability to detect and analyze genetic material. In the initial phase of this method, the DNA sample is subjected to a meticulously controlled cyclic variation of thermal conditions, ultimately leading to the denaturation of double-stranded DNA into single-stranded DNA fragments. Following this critical step, the process involves the strategic annealing of target-specific oligonucleotide primers, which play a pivotal role in the amplification process by enabling the sequential incorporation of deoxyribonucleotides that leads to the synthesis of a complementary DNA strand, thereby resulting in a substantial increase in the number of DNA copies generated. This remarkable capability of PCR not only underscores its utility in various biological and medical research applications but also significantly enhances its sensitivity, allowing for the detection of as few as 10 copies of viral nucleic acid, which is particularly advantageous in diagnostic settings [20].

Loop-Mediated Isothermal Amplification (LAMP)

Loop-mediated isothermal amplification, often referred to as LAMP, is a sophisticated nucleic acid amplification methodology that accomplishes the replication of a specific target sequence through an intricate auto-cycling mechanism of strand-displacement DNA synthesis, which is catalyzed by the DNA polymerase enzyme under isothermal conditions. One of the most significant advantages of this amplification technique is its cost-effectiveness, as it can be conducted using relatively simple equipment such as a water bath or a dry bath apparatus, making it accessible for various laboratory settings and applications [21]. In this innovative method, DNA polymerase operates to facilitate amplification while maintaining strictly regulated isothermal conditions within a temperature range of 60 to 65 degrees Celsius, which is essential for optimal enzymatic activity. Furthermore, the specificity of this assay is greatly enhanced due to the employment of four distinct primers, each designed to recognize and bind to six different target regions within the nucleic acid sequence, thereby ensuring high fidelity in the amplification process [20].

Recombinase Polymerase Amplification Assay (RPA)

In the context of the recombinase polymerase amplification assay, it is important to note that this innovative method does not incorporate the conventional steps of DNA denaturation and primer annealing; rather, the amplification process occurs through the formation of a complex that involves the primer and the recombinase enzyme, which underscores the unique operational mechanisms of this approach. Once this complex is established, it initiates a strand displacement activity within the double-stranded DNA, which results in the amplification of the desired genetic material. Upon completion of the amplification process, the introduction of a probe, which can be either a fluorophore or a quencher, facilitates the real-time detection of the specific DNA fragment of interest, thereby providing immediate feedback on the presence of the target sequence. Notably, this entire amplification process can be executed at a relatively mild temperature range of 37 to 42 degrees Celsius and can be completed in an impressively short duration of approximately 15 minutes, making it a rapid and efficient tool for molecular diagnostics [20].

TREATMENT

The Monkeypox virus is classified within the orthopoxvirus family, which encompasses several viral pathogens that elicit pathogenic conditions akin to those observed in smallpox infections, thereby raising significant concerns regarding public health. A considerable body of research has demonstrated that various antiviral agents exhibit protective mechanisms that are effective against the Monkeypox virus, highlighting the potential for therapeutic intervention in cases of infection. Among the most utilized antiviral drugs in the treatment of orthopoxvirus infections are Cidofovir, Brincidofovir, and Tecovirimat, each of which possesses unique mechanisms of action that contribute to their efficacy in combating these viral pathogens [22].

Cidofovir

Cidofovir has been shown to exhibit optimal therapeutic effectiveness when administered immediately following exposure to the Monkeypox virus, particularly when delivered systemically, which significantly enhances the likelihood of successful health restoration in affected individuals. Clinical observations indicate that patients receiving Cidofovir treatment experience a complete restoration of health coupled with a consistent decline in viral load within a timeframe ranging from 4 to 18 days following the initiation of therapy, thus underscoring its potential as an effective antiviral agent [23]. Cidofovir functions as a monophosphate nucleotide analogue, exerting its pharmacological effects by specifically inhibiting the activity of the DNA polymerase enzyme, which is crucial for viral replication. Presently, Cidofovir is primarily utilized in a topical formulation for the treatment of Monkeypox, although it is important to recognize that this agent is associated with a spectrum of adverse effects, including but not limited to headache, asthenia, fever, skin rash, nausea, vomiting, and ocular abnormalities. The most significant dose-limiting toxicity associated with the administration of Cidofovir is nephrotoxicity, which exhibits a dose-dependent relationship, necessitating careful monitoring of renal function during treatment [24].

Brincidofovir

Brincidofovir, which is classified as a prodrug of Cidofovir, is chemically conjugated to a lipid molecule and has received approval from the Food and Drug Administration (FDA) for its application in the treatment of smallpox disease in adult populations, thereby expanding the therapeutic options available for managing orthopoxvirus infections [25]. This drug operates by inhibiting the viral DNA polymerase enzymes, thereby disrupting the replication of the viral genome and contributing to the overall antiviral effect [26]. The unique lipid component of Brincidofovir closely resembles an endogenous lipid known as lysophosphatidylcholine, which facilitates the entry of the drug into infected cells via natural lipid absorption mechanisms, thus enhancing its bioavailability and therapeutic effectiveness. Once inside the cellular environment, the lipid portion of Brincidofovir undergoes metabolic breakdown, resulting in the release of Cidofovir, which subsequently undergoes intracellular kinase phosphorylation to generate the active form known as Cidofovir diphosphate. However, it is critical to note that the administration of Brincidofovir necessitates continuous monitoring of hepatic function tests, as it has been associated with elevated serum transaminase and bilirubin levels, which may indicate potential hepatotoxicity. Additionally, patients receiving Brincidofovir treatment may experience adverse gastrointestinal effects, including diarrhea and vomiting, which should be considered when evaluating the overall safety profile of this antiviral agent [27].

Tecovirimat

Tecovirimat, which was previously referred to by the designation ST-246, represents a distinctive chemical compound classified as a 4-trifluoromethyl phenol derivative, and it functions as a potent inhibitor of the P37 protein, a molecular entity that is not only highly conserved but also ubiquitously present across all known Orthopoxviruses. The P37 protein engages in critical interactions with various components that are derived from late endosome-originated transport vesicles, thereby facilitating a complex biochemical interaction. This specific molecular interaction ultimately culminates in the assembly of a virus-specific wrapping complex that is essential for the proper encapsulation of enveloped virions. Consequently, it can be inferred that the P37 protein plays a pivotal role in mediating the virulence of these viruses through its indispensable function in the formation of enveloped virions. This intricate mechanism underlines the therapeutic efficacy of Tecovirimat in the treatment of infections caused by orthopoxviruses [24, 28].

FUTURE DIRECTIONS

At present, the viral infection caused by Monkeypox is proliferating at an alarming rate across the globe, thereby establishing itself as a significant and fruitful domain for intensive scientific research and inquiry. Despite the existence of a diverse array of antiviral pharmacological agents that are currently accessible in traditional dosage forms, there exists a compelling opportunity for researchers to explore the potential of these existing drugs by innovatively reformulating them into novel drug

delivery systems that offer numerous advantages when compared to their conventional counterparts. Furthermore, the advancement of sophisticated diagnostic technologies, particularly those that enable rapid and precise testing methodologies, will be instrumental in effectively distinguishing monkeypox from other clinically similar viral diseases, such as smallpox and chickenpox, thereby facilitating timely and accurate diagnosis.

In addition to these advancements, enhanced surveillance teams must be established and mobilized to ensure the early detection and prompt treatment of the disease, particularly in geographical regions characterized by active outbreaks of illness. Moreover, it is of utmost importance to implement a variety of community awareness initiatives and public health campaigns aimed at educating the general populace regarding critical aspects of disease transmission, diagnostic procedures, treatment options, and safety measures, with the overarching goal of significantly reducing the incidence of the disease in affected communities.

CONCLUSION

Monkeypox continues to be recognized as a significant zoonotic disease that poses considerable implications for public health on a global scale. Recent scientific investigations have substantially augmented our understanding of the disease and facilitated the development of improved vaccination strategies.; However, persistent challenges remain, particularly about ensuring equitable access to these essential public health tools for all individuals. Thus, it is evident that ongoing research efforts and robust global cooperation are vital to effectively control the spread of monkeypox and to safeguard the health and well-being of the public at large.

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