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OPHTHALMIC MONKEYPOX: A COMPREHENSIVE REVIEW ON OCULAR INVOLVEMENT, DIAGNOSIS, TREATMENT AND VACCINATION

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ABSTRACT: Ocular monkeypox, a rare but severe consequence of monkeypox virus infection (an Orthopoxvirus), presents with various ophthalmic issues, from conjunctivitis and keratitis to, in severe cases, corneal ulcers and vision loss. Transmission occurs *via* Close contact with sick people, contaminated objects, or respiratory droplets can spread the infection. Among the symptoms include inflamed eyes, swelling, pain, and discharge, often alongside systemic symptoms like fever, rash, and swollen lymph nodes. Prompt diagnosis and treatment, including supportive care, antiviral medications (e.g., tecovirimat or cidofovir), and management of secondary bacterial infections, are crucial to prevent permanent damage. Corneal transplantation may be considered in vision-threatening cases due to corneal damage. Vaccination, especially with the modified vaccinia Ankara (MVA) vaccine, offers protection and lessens the risk of severe complications, including ocular involvement. Public health strategies, like immunization drives and instruction, are vital for outbreak control and minimizing the impact of ocular and systemic monkeypox. More investigation is required to improve and understand the disease's mechanisms and improve treatment, prevention, and surgical approaches.

INTRODUCTION: The monkeypox virus is a double-stranded DNA virus belonging to the Orthopoxvirus genus within the Poxviridae family. It is responsible for monkeypox, a zoonotic illness [1]. This virus primarily resides in tropical forest regions of Central and Western Africa, but infrequent transmission can lead to its spread to other locations. Transmission occurs through various means, including bites or scratches from infected animals, contact with bodily fluids from active lesions, and sexual contact.

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Currently, a global outbreak is underway, with over 35,000 confirmed cases reported across 85 countries [2]. Infected individuals may experience fever, followed by the appearance of a maculopapular rash that evolves into vesicles, pustules, and eventually scabs. Additional symptoms such as swollen lymph nodes and muscle pain may arise within 6 to 13 days, though the incubation period can range from 5 to 21 days.

The illness generally resolves without intervention; however, severe cases have been observed, particularly in children and immunocompromised individuals, with a reported fatality rate of 3% to 6% [3]. Rapid globalization, population migration, and expanding trade systems to contribute the spread of the monkeypox diseases in recent years, leading to outbreaks in many countries worldwide [4-6].

Human-to-human transmission became severe between 1996 and 1997, although the earliest cases were epidemic and recorded in African nations [7, 8]. A few of the modes of transmission include prolonged close contact, respiratory droplets, contaminated personal objects, and direct contact with rash areas [9].

Monkeypox typically differs from smallpox by frequently causing swollen lymph nodes. However, the current outbreak has shown that monkeypox can present in unexpected ways, sometimes without the usual fever or widespread rash. Instead, individuals might develop only a few skin sores that appear at different times. These sores often occur on the genitals, mouth, or rectum areas commonly involved in sexual contact. This association with sexual activity has led to challenges in recognizing and treating monkeypox, resulting in some cases being misdiagnosed or diagnosed late [10].

Additionally, new health complications associated with monkeypox have emerged, such as inflammation of the urethra, rectal discomfort, and difficulty urinating. While the monkeypox virus can potentially be transmitted from mother to unborn child, the specific consequences for pregnant women are still not fully understood [11, 12]. Furthermore, the discovery of the monkeypox virus in semen samples, including instances where the virus remained infectious, has raised concerns about the possibility of sexual transmission [13].

Epidemiology: Monkeypox, a viral illness, was first detected in humans in 1970 in the Democratic Republic of the Congo (DRC) after infecting a nine-month-old infant [14]. While the number of monkeypox outbreaks has increased since then, they have primarily been confined to the African continent, with limited spread to North America and Europe [15]. Early outbreaks were relatively small, between 1970 and 1979, 48 confirmed cases were recorded in six African countries. By1986, the number of human cases had risen to over 400, with a concerning mortality rate of approximately 10%. Monkeypox outbreaks continue to occur in equatorial Central and West Africa [16], particularly in the DRC. Between1991 and 1999 [17], over 500 cases were reported in the DRC alone. The Congo Basin within the DRC remains an endemic region for monkeypox, with a high case fatality rate. Currently [18], an estimated 1,000 cases are reported annually in the DRC.

Viral Re-Emergence: The global outbreak of monkeypox (MPV) became apparent in May 2022. On May 18, Portugal, Spain, and Canada reported 14, 7, and 13 cases, respectively [19]. The next day, Belgium, Sweden, and Italy confirmed their first infections. Australia identified two cases on May 20, both linked to recent travel from Europe. That same day, France, Germany, and the Netherlands also detected their first cases. Meanwhile, the UK reported 11 additional cases on May 20, raising its total to 71. Belgium became the first nation to enforce a 21-day isolation period for MPV [20]. On May 21, Israel and Switzerland confirmed their initial cases. By June 3, Spain, which had originally reported cases on May 18, experienced a sharp rise of 20 new infections, bringing its total to 186 [21].

The global spread of monkeypox continued to accelerate in late May 2022. Denmark reported first case on May 23, linked to travel from Canary Islands. On May 24, Quebec, Canada, confirmed 15 cases, and the Czech Republic reported its first case, linked to an international music festival held in Belgium. By May 24, monkeypox cases had been reported in 19 countries worldwide. The source of the outbreak remained unknown, but the emerging nature of the outbreak suggested both human-to-human and possibly animal-to-human transmission. In late May, the United Arab Emirates first case was reported in a 29-year-old woman who had recently traveled from West Africa. These developments further highlighted the rapid global spread of monkeypox beyond its traditionally endemic regions in Africa.

Manifestations of Viral Infections: Monkeypox infection shares some similarities with milder cases of smallpox [22], but a key distinction lies in the presence of swollen lymph nodes (lymphadenopathy), which is not observed in smallpox. Initial symptoms typically include fever, headache, backache, muscle aches, and fatigue. The incubation period for monkeypox generally lasts between 7 and 14 days, though it can extend up to 21 days. Symptoms usually begin with fever, followed by the appearance of a facial rash that gradually spreads to other parts of the body.

Lesions typically first appear in the oropharynx (back of the throat) before progressing further. Serum antibodies become detectable approximately two weeks after exposure to the virus [23]. The mortality rate of monkeypox varies significantly, ranging from 1% to 10%, depending on the specific strain (clade) of the virus and the availability of adequate medical care [24].

Viral Entry and the Development of Disease: The monkeypox virus (MPV) can enter the human body through the nasopharynx, oropharynx, or direct skin contact. After entry, the disease replicates at the initial position of infection already spreading to adjacent lymph nodes. It then disseminates to other organs via the bloodstream.

MPV, like other orthopoxviruses, it has an oval or rectangular structure surrounded by a lipoprotein outer membrane [25]. Its genome consists of 197 kilobases of linear, DNA has twin strands. Despite being a DNA virus, MPV reproduces in the cytoplasm of the host cell. Viral repetition, transcription, and assembly of new virus-related particles require the synthesis of numerous viral proteins [26]. Poxviruses, including MPV, can enter host cells through various mechanisms, including fusion, endocytosis, and macropinocytosis [27].

The monkeypox virus has two main clades: the West African clade, which is generally milder, and the Congo Basin clade, which originated in Central Africa and is more severe. Recent genetic analysis of monkeypox virus samples in Europe suggests that they closely resemble the West African clade [28], indicative of a potentially less severemethod of the illness. However, further research is needed to confirm this finding [29]. The 2003 U.S. outbreak, which involved the West African clade, highlights that the cruelty of the illness can vary between different viral clades. In general, both human and non-human primate infections caused by the West African clade tend to be less severe compared to those caused by the Congo Basin clade [30, 31].

Monkeypox is known to trigger an immune response involving T-cells [32]. However, studies have shown that T-cells from individuals previously infected with monkeypox exhibit a limited inflammatory response. This suggests that the monkeypox virus might produce a factor that suppresses the body's T-cell response [33], potentially enhancing its ability to cause infection. A crucial immune-modulating gene, which suppresses complement enzymes, is found in the Central African clade of the monkeypox virus but is absent in the West African clade. This gene's presence may play a role in the greater virulence observed in the Central African strain compared to its West African counterpart [34, 35]. The Central African monkeypox clade also exhibits a greater ability to manipulate host cell death (apoptosis), suggesting a stronger suppression of host immune responses than the West African clade [36].

Genomic comparisons revealed a nucleotide variation of approximately 0.55 to 0.56% between three West African monkeypox strains (SL-V70, COP-58, and WRAIR-61) and a Central African strain (ZAI-96). Despite this difference, the West African strain is estimated to have 171 unique genes, whereas the Central African strain is characterized by 173 distinct and well-defined genes.Ananalysis of 56 virulence-related genes, selected due to their role in pathogenic differences between the two clades, revealed that 53 were common to both strains. The greatest genetic variations among the strains were identified in the orthologs of BR-203, BR-209, and COP-C3L [37].

Eye: Corneal scarring and vision loss are serious complications of monkeypox infection [38]. Optical complications remain arenot unique to monkeypox; they stood also reported in 5–9% of smallpox (variola virus) infections. Before the eradication of smallpox, preventative measures to reduce blindness included eye lubrication for at-risk individuals and vitamin supplementation. These interventions were particularly crucial for preventing secondary bacterial corneal infections, which often developed later in the disease course. In smallpox, severe ocular damage was frequently linked to bacterial superinfection of corneal ulcers, leading to serious complications such as corneal rupture, anterior staphyloma, and bulbar phthisis [39]. Topical trifluridine has been used in several cases to accelerate symptom relief and reduce long-term corneal scarring. Trifluridine is regarded as the primary treatment for ocular vaccinia, while topical or oral antibiotics may be used to manage bacterial superinfection or as a preventive measure [40–42].

Nine months after the original wound had healed, a recurrent corneal erosion caused by a cowpox virus poison was informed (i.e., in the absence of culturable virus). In this patient, prolonged corneal damage and viral persistence were believed to have resulted from the administration of steroid eye drops for inflammation management. She eventually underwent multiple corneal transplants.

In earlier cases, long-term vision loss, corneal opacity, and tissue scarring were observed. Over several years of follow-up, monkeypox patients with ocular complications reported persistent discomfort (personal communication, CDC, A. McCollum).During the smallpox era, twno relatively simple interventions topical antibiotics and improved eye lubrication were used to treat ocular complications. These methods may also be beneficial in monkeypox-related eye infections. To prevent long-term vision impairment, early and targeted treatment aimed at supporting viral clearance similar to approaches used for ocular vaccinia may be advantageous [43].

Differential Diagnosis of Monkeypox: Apart from MPXV, several other orthopoxviruses and their associated vaccinations can cause similar corneal infections and complications. Among the poxviruses, smallpox has been extensively studied. A key distinguishing feature of MPXV is lymphadenopathy, which sets it apart from smallpox and chickenpox infections. This symptom usually appears in the early stages of the disease [44]. It has been reported that 5–9% of smallpox patients experience ocular symptoms. Based on these similarities, corneal ulceration is the most common ocular complication of MPXV, posing a significant risk to vision through various mechanisms, including perforation and endophthalmitis [45]. Patients who develop pustular lesions on the eyelids or conjunctiva after receiving a smallpox vaccination or recently coming into contact with a vaccinated individual should be suspected of having ocular vaccinia [46, 47]. Accidental eye rubbing after touching the smallpox vaccination site can lead to blepharoconjunctivitis, either through self-inoculation (autoinoculation) or close contact [48]. Approximately 10 to 20 cases of ocular vaccinia occur per million primary smallpox vaccinations, and the condition is associated with more severe complications in first-time vaccine recipients than in those who are revaccinated. The risk of ocular vaccinia was estimated to be similar for both standard smallpox vaccines Dryvax and ACAM2000 [49]. However, the recently approved MPXV vaccines, (MVA-BN) Modified Vaccinia Ankara-Bavarian Nordic, as well as the non-replicating smallpox vaccine, do not cause ocular vaccinia.

Eye infections caused by orthopoxviruses can resemble those caused by other viruses, such as Molluscumcontagiosum (MC), Varicella-Zoster virus and Herpes simplex virus. While monkeypox virus (MPXV) can sometimes lead to blepharoconjunctivitis (inflammation of the eyelids and conjunctiva), most individuals ill with Herpes Simplex Virus type 1 (HSV-1) which can potentially cause similar ocular symptoms, such as superficial punctate keratitis (SPK) remain asymptomatic [50].

Additionally, HSV ocular infections can present in three primary forms: retinitis, uveitis, and keratitis. Diagnosing herpes zoster ophthalmicus, which results from the reactivation of latent VZV, requires a history of a prior rash or noticeable skin lesions [51]. This condition can cause frontal corneal infiltrates, epithelial keratitis, and conjunctivitis, mimicking ocular manifestations of poxvirus infections [52.53].

Ocular Mpox:

Epidemiology: The collective term "Mpox-related ophthalmic diseases (MPXROD)" refers to ocular defects or symptoms associated with Mpox. The usual ocular manifestations of Mpox include conjunctivitis, pustules on the eyelids, and other eye-related complications. The world-wide spread of the Clade IIb serotype led to the 2022 MPXV pandemic, whereas Clades I and IIa of the virus are primarily found and transmitted within Africa [54].

Compared to the 2022 pandemic, MPXROD-related infections were more prevalentthrough the previous Clade I epidemic [55, 56]. Ocular symptoms of MPXV can present in various forms, often caused by autoinoculation of the eye. However, these symptoms are commonlyreflected a less frequent manifestation of the sickness [57]. A education conducted on 185 Mpox-infected patients in Paris revealed that 1% developed eye-

related complications [58]. Conjunctivitis, along with other symptoms, was a cause of hospitalization, and some patients presented pustules on the eyelids in addition to conjunctivitis. Similarly, studies conducted in the Democratic Republic of the Congo also informedocular complications [59]. Notably, when Mpox patients developed conjunctivitis, a significant proportion (47%) became bedridden (Figure A), compared to only 16% of those without conjunctivitis. This suggests a potential link between severe Mpox infection and conjunctivitis. Further research is needed to understand how Mpox targets ocular cells and what factors contribute to eye-related symptoms [60].

Ocular Surface Complications: Although Mpox symptoms in the eye's after region are quite uncommon, The ocular surface is usually affected by ocular mpox, and at the moment of release .Clinical pictures of ocular Mpox include the classic signs of epithelial keratitis, anterior uveitis, significant conjunctivitis, and conjunctival erosions, along with geographical corneal ulcers that progress into Spartan corneal thinning, eyelid papules, peripheral corneal semi-lunar gain access to, and corneal opacification [61].

FIG. 1: KERATITIS, CORNEAL OPACITIES, AND DIFFUSE OCULAR HYPEREMIA[62]

Conjunctivitis: Bacterial or viral infections are the primary causes of pink eye (conjunctivitis), particularly in child and the elder people, due in part to their weakened immune systems. In such cases, an allergic response often occurs, accompanied by a noticeable mucus discharge from the eye. In nearly 20% of Mpox cases, conjunctivitis has been the most frequently reported symptom. Patients diagnosed with conjunctivitis in Mpox also exhibited higher rates of added symptoms, including chills, nausea, sweating, mouth ulcers, sore throat, general malaise, lymphadenopathy, and photophobia. Blepharo-conjunctivitis is another possible ocular complication of Mpox, characterized by inflammation affecting both the eyelid and the conjunctiva [63]. Clinical investigations suggest that MPXV has the the capacity to cause inflammation and infect conjunctival epithelial cells.

Keratitis: MPXV infection has been linked to several cases of keratitis, an inflammation of the cornea, emphasizing the virus's potential to cause significant ocular damage [64]. One reported case involved a patient with a corneal epithelial defect, discomfort, corneal edema, and keratic precipitates, indicating a corneal infection [65].

Keratitis is a leading cause of blindness resulting from various infections. While ocular epithelial cells help defend against pathogens by releasing immune mediators, excessive immune responses can lead to corneal scarring. Interestingly, studies in mice suggest that the absence of certain immune cells (CD4+ and CD8+ T cells) can worsen keratitis caused by a related virus [66]. The mechanisms behind MPXV-induced keratitis remain unclear, but given its potential to cause vision loss, further research is crucial to identify the viral and host factors involved.

Blepharitis and Periorbital Lesions: Eyelid inflammation, or blepharitis, affects the margins of both eyelids and often occurs when small oil glands are based on eyelashes become closed, leading to redness and discomfort. In individuals with Mpox, MPXV have been related to blepharitis. According to reports, blepharitis is more prevalent in unvaccinated Mpox patients (30%) compared to those who previously received a smallpox vaccination (7%) [67].

MPXV has also been associated with various eye-related warning sign, excluding conjunctivitis, blepharoconjunctivitis, and keratitis, particularly affecting the periorbital area and eyelids [68]. Additionally, there are reports of periorbital rashes in MPXV patients. These rashes, along with further facial lesions, may not only indicate direct viral transmission through skin contact but also suggest a potential risk of viral sexual transmission. Furthermore, these symptoms often occur alongside systemic signs such as severe cervical lymphadenopathy and fever [69].

Host Factors Contributing to Ocular Mpox Development: Glycosaminoglycans (GAGs) and proteoglycans serve as host receptors for MPXV, facilitating viral entry and interaction with key signaling pathways, including TGF-β, FAK, and ERK [70, 71]. Among these, the TGF-β pathway plays a critical starring role in optical surface homeostasis then disease progression, as it is expressed by stromal keratocytes, conjunctival, corneal, and limbal epithelial cells [72].

This pathway is particularly essential for enhancing re-epithelialization, promoting epidermal growth factor receptor (EGFR) recycling, and accelerating wound healing [73, 74]. Additionally, it supports the formation of the corneal epithelial cell adhesion complex, increases protease inhibitor expression, and recruits neutrophils and monocytes during inflammation [75, 76].

During conjunctivitis, the smallpox virus has been shown to replicate aggressively, triggering the host to produce epidermal growth factor (EGF) and other genes that enhance cell tropism and pathogenicity. Since MPXV can be spread through tears, this may explain the development of ocular symptoms.

Additionally, poxviral DNA encodes several immune-evasive proteins, such as MC132, p28 ubiquitin ligase, and histone deacetylase proteins, which help the virus evade the host's innate antiviral immune response. This immune evasion more promotes the formation of skin wounds[77].Much of our present-day understanding of MPXV-host interactions comes from studies on related viruses, but the mechanisms underlying MPXV ocular pathogenesis remain largely unexplored. Further research is needed to uncover the host factors that MPXV exploits or suppresses in the development of ocular disease [78].

Transmission: The term "zoonotic transmission" refers to the transfer of illness from animals to humans or between individuals [79]. Primary zoonotic transmission occurs when humans come into direct contact with or consume infected natural hosts. Body fluids, respiratory droplets, or skin lesions from an infected person can all result in secondary transmission [80]. A study found that 95% of infected patients reported intimate sexual contact, and males who identified as gay or bisexual made over 98% of the affected population. Lesions most frequently occur in the anogenital area, further suggesting that sexual contact may be a key transmission route[81–82]. However, sexual transmission has not yet been fully confirmed as a distinct infection pathway, and further research is ongoing [83].

Kaler *et al.* emphasize that respiratory droplets remain the primary mode of human-to-human transmission. Additionally, children and immune-compromised persons such as those with AIDS or undertaking antiretroviral therapy are at a higher risk of severe infection and complications[84].

Ophthalmic Manifestations: In addition to systemic symptoms, ocular involvement in mpox can present with eye pain, redness, excessive tearing, photophobia, discharge, periorbital swelling, and impaired vision. Research indicates that unvaccinated individuals (74%) are more likely to experience ocular complications compared to those who have received a smallpox vaccination (39.5%).

Mpox has been shown to affect multiple ocular structures, including the sclera, cornea, conjunctiva, and eyelids. Kaufman & Co[85] introduced the term "Mpox-Related Ophthalmic Disease" (MPOXROD) to describe the wide range of ocular symptoms associated with mpox infection [86].

Treatment: Supportive care is the primary approach for managing systemic monkeypox infections, as MPXV infection typically resolves on its own[87–88]. However, patients with severe illness or those at risk of complications may require systemic antiviral treatment. The available antiviral options include tecovirimat, cidofovir, brincidofovir, and vaccinia immune globulin intravenous (VIGIV) [89–90]. Tecovirimat suppresses the Orthopoxvirus F13L gene's product. In 2018, tecovirimat was licensed by the U.S. FDA to treat smallpox, primarily based on animal research[91]. The CDC has a process for providing access to investigational drugs (those not yet fully approved) for treating non-variolaorthopoxvirus infections, including monkeypox. Because monkeypox can affect the eyes and the eye is considered a "special hazard" area, tecovirimat may be considered a

treatment option [92]. However, it remains unclear how effective tecovirimat is for MPXROD and how bioavailable it is in the eyes [93–94].To treat cytomegalovirus retinitis, the FDA has approved cidofovir, a gene nucleotide analog that blocks viral DNA polymerase. In vitro as well as in animal models, it has demonstrated effectiveness against orthopoxviruses, including MPXV. In 2021, the FDA approved brincidofovir, a cidofovirprodrug, for the treatment of smallpox following research in animals and in-vitro, which demonstrated its efficacy against orthopoxviruses.

Orthopoxvirus species. While brincidofovir is not currently part of the national medical stockpile, the CDC is developing a program to allow its use in treating monkeypox. VIGIV is derived from purified plasma immune globulin, obtained from donors who have been vaccinated and possess high levels of anti-vacciniatiters. It is FDA-approved for treating complications related to vaccinia vaccination; however, it can also be used to manage other orthopoxvirus infections through an expanded-access program. In addition to benefiting individuals with significant T-cell depletion (such as those with HIV/AIDS), VIGIV can be administered for severe monkeypox infections or as post-exposure prophylaxis [95].

In a vaccinia keratitis animal model, VIGIV therapywas linked to a rise in corneal scarring [96]. However, further trainings are not confirmed this correlation. Although the CDC advises against using VIGIV when monkeypox is accompanied by active Keratitis doesn't seem to be a precise reason not to use VIGIV [97]. Currently, there is no approved antiviral topical treatment for monkeypox infections. The only reported case of ocular vaccinia utilized the FDA-approved1% trifluridine ophthalmic solution, which is primarily used to treat herpes simplex virus keratitis [98]. This medication has also demonstrated in vitro effectiveness against orthopoxviruses [99].

There is currently limited clinical knowledge on trifluridine's efficacy in treating human MPXROD [100]. In cases of MPXROD, the CDC recommends considering topical trifluridine. Historically, MPXROD and other ophthalmic symptoms of Orthopoxvirus infections have been managed with extensive topical lubrication [101], which remains a crucial part of supportive ocular treatment. Topical antibiotics may also play a key role in preventing bacterial superinfection, particularly in cases involving corneal ulcers and epithelial abnormalities [102, 103].

Vaccination: Although there is currently no vaccine specifically designed to prevent monkeypox, smallpox immunization may provide cross-protection against monkeypox, with an efficacy of up to 85% [104]. However, since the 1970s, the eradication of smallpox has led to the discontinuation of routine smallpox vaccination in the United States. Furthermore, the extent to which a smallpox vaccine administered decades ago provides protection against monkeypox remains uncertain. As previously mentioned, epidemic data suggest that prior immunization is associated with lower MPXROD rates [105-106]. However, it remains unclear whether an extended interval between vaccinations may weaken this protective association [107].

Vaccination against smallpox is currently being utilized to prevent the spread of monkeypox. The currently available smallpox vaccines include JYNNEOS and ACAM2000. ACAM2000 utilizes a replication-competent vaccine virus and is delivered through skin scarification. On the other hand, JYNNEOS is given subcutaneously in two doses and contains a modified Ankara vaccinia strain with limited replication ability [108]. These vaccines are not widely accessible to the general public and are primarily reserved for post-exposure prophylaxis (PEP) and pre-exposure prophylaxis (PrEP) in select high-risk occupational groups. Theseinclude laboratory personnel handling orthopoxviruses, research workers, and select public health and healthcare professionals designated by public health authorities [109-110].

Clinicians should stay updated on vaccine recommendations, as eligibility criteria continue to expand based on vaccine availability and epidemic trends. Local public health agencies provide the latest vaccine distribution updates, and JYNNEOS is becoming increasingly accessible [111, 112].

Infection Risk from Corneal Transplants: At present, no cases of monkeypox transmission have been associated with corneal transplants. However,

as noted earlier, individuals experiencing active monkeypox conjunctivitis may still release the virus [113–115]. A monkeypox outbreak presents a unique challenge, as corneal transplants are far more common in wealthy nations, where the disease remains relatively rare. In July 2022, the Eye Bank Connotation of America introduced protective measures to reduce the risk of MPXV transmission through corneal transplants from cadavers. Considering the 5–21 day MPXV incubation period, the association recommends excluding donors who meet any of the following criteria: MPXV-positive status, Unexplained Orthopoxvirus positivity, Presence of a monkeypox-related rash, close connection with a confirmed monkeypox case within the past 21 days [116] Cornea specialists should stay informed about updates from the Eye Bank Connotation of America as the outburst continues to evolve [117].

CONCLUSION: Monkeypox affecting the eyes is a serious complication that can threaten vision. It can cause various eye problems, from conjunctivitis and eyelid inflammation to corneal ulcers and, in severe cases, blindness. Prompt diagnosis and treatment are crucial to prevent permanent damage. Treatment involves antiviral medications, supportive care, and management of any secondary infections. Surgery, like corneal transplantation, might be necessary in severe cases. Vaccination, especially with the MVA vaccine, is a key preventative measure that can greatly reduce the risk and severity of monkeypox, including its effects on the eyes. Public health inventiveness, such as vaccination programs and educational campaigns, are essential for controlling the virus's spread. Continued research is vital for improving our understanding of the disease, refining treatments and surgical techniques, and developing better prevention strategies to protect both eye health and overall well-being.

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