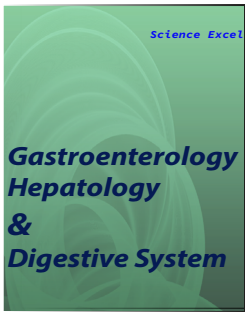
Gastroenterology Hepatology and Digestive System

Case Report



Monkeypox

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Introduction

The first case of Monkeypox in humans was described in 1970 in a 9-month-old child in the Democratic Republic of Congo. Thereafter, transmission of the virus was contained by mass vaccination for Smallpox (smallpox), which conferred cross-immunity to Monkeypox infection in vaccinated populations. The central and western regions of the African continent are endemic areas of the disease, a condition related to the proximity of local populations to wild animals that are the most common reservoirs of the virus.

Despite this, the incidence of the disease has grown exponentially in the last two decades, accounting for more cases in this period than in the entire time of knowledge of the disease, 45 years. In May 2022, the start of a new Monkeypox outbreak overlapped with the end of the COVID-19 pandemic. So far, the WHO has counted 41,664 cases distributed in 96 countries, mainly in Africa, the Americas and Europe.

The medical community was surprised not only by the return of a disease long limited to endemic areas, but by the unprecedented variety of signs and symptoms, among which anal manifestations are rarely described in the literature. Considering the locations of occurrence of lesions, the genital region is the one with the highest frequency of occurrence of lesions (60.7%). This article presents the case of a 33-year-old patient diagnosed with human Monkeypox in São Paulo, Brazil, in August 2022. We also describe a comparison of the pathognomonic manifestations and lesions of monkeypox with those found in the new outbreak, especially the rectal ones. and annals.

Case report

A 33-year-old man was urgently admitted for 5 days of acute anal pain associated with anal and perianal ulcers with vesiculopustular eruptions.

The patient reports being a carrier of the human immunodeficiency virus (HIV) with clinical control under follow-up with an infectious disease physician undergoing treatment using antiretroviral drugs (ART) with negative viral load to date.

He denied any other clinical history or previous use of other medications in addition to those he was habitually used. He sought medical attention 3 days ago being diagnosed with genital herpes and treated with topical acyclovir without clinical improvement.

He reports that after the 4th day, he presented clinical worsening of anal pain with worsening of skin lesions and began with mucopurulent anal secretion associated with unmeasured fever, sweating and skin lesions in the penile region (similar to anal lesions) and myalgia. One day ago, he presented with diffuse vesicular lesions in the upper limbs, chest and lower limbs, and then sought emergency care.

During admission, he mentioned that he was a resident of the central area of São Paulo, single and that he had not traveled recently to endemic areas. She reports that she had sex with 5 partners on the same day for 10 days without using a condom. He denied using illicit drugs and reported using alcohol sporadically.

On examination, the temperature was 36.9ºC, blood pressure was 129x72mmHg and saturation was 98% on room air. There were 4 perianal ulcers up to 0.5 cm in

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diameter with raised edges and extremely painful to the touch. In addition, there were numerous papulovesicular lesions in the region of the upper and lower limbs, as well as the thorax and posterior surface of the trunk, measuring up to 3 mm in diameter, filled with clear and transparent fluid, with discrete erythema in between.

Laboratory tests were initially not normal. A rapid HIV test was performed, which was confirmed positive and tests for other sexually transmitted diseases were negative, as well as other serologies of acute viral diseases. Anal pain did not show complete clinical improvement with the use of common analgesics and opioids. An evaluation of the coloproctology was requested to perform an anoscopy under narcosis, which showed an area of rectal and anal enanthema with the presence of superficial ulcerations and purulent exudate associated with intense proctitis at the site.

The possibility of diagnosing monkeypox (poxvirus) was raised due to the existence of the outbreak in other countries and the existence of cases in Brazil. A PCR examination of an anal skin lesion swab was requested, analyzed by the Adolf Lutz Institute, confirming the existence of Monkeypox virus in the sample sent.

Discussion

The first case of Monkeypox was identified in 1958 in Cynomolgus monkeys transported from Africa to Copenhagen, Denmark, for research purposes, hence the name of the disease as monkeypox. Historically, the disease was first reported in 1970 in Central Africa, affecting more countries in marginalized contexts. During the smallpox peak, there were no reports of monkeypox cases, which can be justified by the great similarity between the clinical picture of the diseases and the focus being on smallpox, leading to a blurring of the distinction between the two, in addition to the lack of laboratory confirmation of the disease. etiological agent. In 1980, with the smallpox vaccination campaign, the World Health Assembly declared the disease eradicated, and routine vaccination was interrupted, in addition, data show that vaccination brought 85% protection against monkeypox, being those unvaccinated people account for about 80-96% of monkeypox cases [1,2]..

Epidemiology

The first cases of monkeypox were recorded in Africa between 1970 and 1979, with the Dominican Republic of Congo being the most affected country, followed by Nigeria. In 2003, the first cases outside Africa (USA) were recorded, in which 53 people aged between 4 and 53 were affected. Most reported cases had no history of smallpox vaccination [3].

On May 6, 2022, a case of monkeypox was registered in the United Kingdom whose individual had traveled to Nigeria. Since then, the number of cases outside Africa has grown sharply, causing the World Health Organization to consider it a public health emergency [4].

A survey carried out by showed that 98% of those infected since the beginning of May were gay and bisexual men, that is, men who have sex with men (MSM) and 75% were white. Of these 98% infected, 41% were living with HIV in a controlled manner, and most of those who had the infection were undergoing antiretroviral therapy. In addition, the average age of infected people was 38 years [4].

Etiology

Monkeypox virus (MPV), responsible for monkeypox disease, belongs to the Poxviridae family, taxonomically classified in the subfamilies: Entomopoxvirinae, characterizing the viruses with potential to infect insects, and Chordopoxvirinae, with those capable of infecting vertebrates, such as MPV. The main hosts of the viruses belonging to the Poxviridae family are not humans, but rodents, rabbits, and primates, which can transmit the virus to humans. Also, MPV belongs to the genus Orthopoxvirus [1,5-10].

Viruses belonging to this genus, as is the case of MPV, have a set of proteins encoded by virulence genes that act as modulators when in contact with the host immune system components. After analyzing the genomic sequence of MPV from cases diagnosed in West and Central Africa and the cases related to the outbreak that occurred in the United States in 2003, it was observed the existence of two variants, West African and Central African or Congo Basin, the latter being more virulent and responsible for more severe cases [5,8,9].

The smallpox virus Orthopoxvirus variolae is closely related to MPV because they belong to the same genus, presenting clinical similarities. Thus, hypotheses have been raised in an attempt to explain the resurgence of cases of monkeypox. The hypotheses of decreased immunity in the population, which could contribute to the dissipation of cases, and of viral genetic evolution prevail. However, deforestation could be contributing to transmission from animals to humans [2].

Animal-to-human transmission is defined as direct contact from exposure to infected animals, such as rodents, rabbits, and non-human primates, most commonly from body fluids, such as saliva, respiratory secretions, and exudate from skin or mucosal lesions. Viral shedding through feces may represent another source of exposure. Human-to-human transmission can involve exposure to respiratory droplets, contact with lesions of an infected individual, contact with contaminated objects/surfaces, or through unprotected sexual intercourse. Thus, reports show that such transmission has occurred through household and nosocomial contacts [5,8,11].

Pathophysiology

The definitive animal reservoir of Monkeypox virus (MVP) has not yet been clarified, however the virus has been isolated from several animal species, including small mammals and non-human primates. In reported cases where MVP was isolated from wild animals, the animals demonstrated smallpox-like lesions consistent with active infection. It is unknown whether asymptomatic transport of MVP occurs in the animal reservoir [3,5].

Monkeypox virus is an enveloped, double-stranded, DNA virus having approximately 200,000 base pairs. Poxviruses rely heavily on virus-encoded proteins that allow them to replicate in the cytoplasm. The central part of the genome contains genes involved in key functions such as transcription and virus

assembly, while those located at the terminals are involved in virus-host interactions. The larger size of the orthopoxvirus alerts the individual's immune system very early and thus generates an immune response very easily [6,11].

To evade the host's immune system, orthopoxviruses are equipped with a set of proteins encoded by virulence genes that will act as modulators when directed against components of the host's immune system. These proteins are categorized into two groups according to their intracellular or extracellular functioning, which only differ in the expression of surface glycoproteins [5,11].

Within intracellular functioning, viro transducer proteins act by interfering with the cell's ability to respond to infection, including oxidative burst and apoptotic pathways. Virostealth proteins reduce the detection of the virus by the host immune system through the down regulation of immune recognition molecules such as major class 1 histocompatibility complex (MHC 1) and TCD+4 [5,6,11]. In the extracellular part, viromimic proteins modulate the immune system response. Viroreceptors are secreted or present as cell surface glycoproteins that competitively bind to host cytokines and chemokines. Virokines form viral mimics of host cytokines, chemokines, and growth factors that are effective in both subverting host responses [5,6,7,11].

Glycosaminoglycans, which are ubiquitously expressed on the surface of mammalian cells, are considered crucial for virion binding to the cell membrane, although cell receptors have not been fully characterized [6].

Thus, after entry, the monkeypox virus replicates at the site of inoculation, and after replication, in primary viremia, the viral load spreads to the local lymph nodes. In secondary viremia, the viral load will reach distant lymph nodes and organs through the circulation. This entire process represents the incubation period, normally lasting from 7 to 14 days with an upper limit of 21 days. Clinical manifestation is not visible during the incubation phase and therefore the incubation period is not contagious. These symptoms may be correlated with the prodromal stage [5,11].

During the prodromal stage, secondary viremia occurs from Organs lymphoid organs to the skin and tertiary organs such as the lungs, eyes, gastrointestinal tract and it is during this prodromal state that an individual is considered the most infectious [7,11].

Clinical manifestations

Most clinical features of Monkeypox infection mirror those of smallpox. Both diseases present themselves with papular lesions and ulcers. The most frequent systemic features of Monkeypox symptoms include fever (62%), lymphadenopathy (56%), lethargy (41%), myalgia (31%), and headache (27%), symptoms that often precede a generalized rash [3 ,4].

Prodromal symptoms of MPV develop up to 4 days after exposure [3,4,12,13], being called “prodromes” because they manifest before the rash, preceding it by one day [5,11]. These include flu-like illness (fever, chills, sore throat, cough, asia, diarrhea, nausea and vomiting); lymphadenopathy (a distinctive feature of Smallpox) and enanthem, often in the mouth and anus. However, in the current MPV (2022 identified as the Africa clade), typical prodrome symptoms were not observed or often after the rash outbreak [5].

The initial febrile prodrome is accompanied by generalized headache and fatigue. Fever is usually limited, disappearing within 0 to 5 days after the rash starts. The occurrence of a second febrile period, that occurs when the skin lesions become pustular, has been associated with the deterioration of the patient's general condition [3,4,12].

Prior to or with the development of rashes, unilateral/bilateral maxillary, cervical or inguinal lymphadenopathy (1-4 cm in diameter) is present in many patients. Enlarged lymph nodes are firm [3,4,12]. There is a hypothesis that the presence of adenopathy may be an indication that there is immunological recognition and a more effective response to monkeypox virus infection compared to smallpox virus [12]. Although the route of infection can influence the clinical manifestations, initial skin lesions usually present in the anogenital region and then appear on the face, palms of the hands and soles of the feet and only then spread centrifugally throughout the body, lasting 2 to 4 weeks [3,4,5,13]. Skin lesions progress through macular, papular, vesicular and pustular stages, followed by healing crusts. In addition, the lesions present at the same stage of development in a particular region of the body [13]. The number of lesions in a given patient can vary from a few to thousands [3,4,13]. The extensive skin disturbance raises concerns about secondary bacterial infections of the skin, being seen in 19% of unvaccinated MPV patients [12].

Lesions are often seen in the oral cavity, including pharyngitis, odynophagia, epiglottitis, and oral or tonsillar lesions, which can cause difficulties in drinking and eating [7]. In addition, vomiting or diarrhea may occur in the second week of illness, contributing to severe dehydration [5,12].

A 2022 international multicenter case series study, which included a total of 528 confirmed cases of Monkeypox, reported that skin lesions were observed in 95% of patients, with the most common anatomic sites being the anogenital region (73%); torso, arms or legs (55%); face (25%); palms and soles of the feet (10%). A total of 54 patients had only a single genital ulcer, which highlights the potential for misdiagnosis as a different STD [7]. Anorectal mucosal involvement was reported as a presenting symptom in 11.5% of patients. This involvement has been associated with anorectal pain, proctitis, tenesmus or diarrhea or a combination of these symptoms. Severe anorectal pain due to anorectal lesions was characteristic in these patients with anal manifestation [3,4,9,14,15].

Diagnosis

The diagnosis of current Monkeypox infection is based on clinical signs and symptoms, history of recent contact with people diagnosed or suspected of having Monkeypox, contact with person with suspected rashes, multiple or anonymous sexual partners lately, contact with animals known to be Monkeypox reservoirs or any infected surface or fomites and real-time polymerase chain reaction (PCR) test [6,12,13].

A very detailed history should be taken from patients. It’s

important to collect information regarding sleeping, living, drinking and eating habits. The absence of travel history or a close contact with a rash or with suspected or confirmed Monkeypox infection should not exclude the possibility of this diagnosis. A very careful skin examination should be performed. Clinicians should be aware of the atypical presentations of the infection described in the ongoing 2022 outbreak. [6]

The real time PCR molecular test is the most accurate laboratory analysis for Monkeypox confirmation for patients with clinical symptoms and epidemiological information. This test is highly sensitive and can efficiently detect viral DNA. Skin samples should be taken to confirm the infection [5,6,9,12,16]. Swabs of vesicular lesions, exudate, or crusts stored in a dry, sterile tube are the best samples for the laboratory analysis for its simplicity in obtain and analyze [12,16]. The most indicate, if possible, is the obtaining of more than one sample from two separate open lesions on different parts of the body. The open lesions are more adequate to collect sample secretions containing viruses [6]. Other laboratory tests could be taken such as anti-orthopoxvirus IgG, anti-orthopoxvirus IgM, Immunochemistry tests but they are not Monkeypox specific, cell culture provides virus strains for further characterization, but it is restricted to accredited biosafety level 3 reference laboratories and is less sensitive but can aid in confirmation [5,6,9,12,16]. Monkeypox serology can cross-react with smallpox vaccination so the results should take this into consideration and analyze each case [6]. The genetic material of the virus could be also retrieved in blood, urine, upper respiratory tract and seminal fluid [9].

Patients could be categorized in to three categories: suspect case when they present localized or generalized rash and/or anogenital manifestations (i.e. pruritus, ulcers) with sudden one or more than these symptoms: fever (≥38.0°C), asthenia, myalgia, backache, headache, lymphadenopathy; probable case when patients, in addition to the previous criteria, presents also one or more of the ensuing conditions: contact with a suspected, probable, or confirmed Monkeypox case within 21 days before the onset of symptoms, sexual intercourse with multiple or anonymous random sexual partners within 21 days before the symptom onset, hospitalization due to a clinical condition consistent with a suspected case, and travel history to Monkeypox endemic countries within 21 days before the symptom onset; confirmed case when patients have a positive reliable and validated laboratory test for Monkeypox infection, such as real-time PCR test [7,13,15].

Monkeypox can manifest similar to many other diseases that is why is important to rule out differential diagnosis before its confirmation. Diseases like sexually transmitted diseases such as syphilis, disseminated gonorrhea, condyloma accuminata, acute HIV; diseases caused by other virus such as varicella zoster, herpes simplex, chickenpox; other orthopoxvirus such as molluscum contagiosum, smallpox, pseudocowpox may resemble or even co-infect with Monkeypox [5,7,9,13].

Treatment

The infection has a mild and self-limited course of the disease, without the need for specific antiviral therapy. In mild cases, supportive care is required with attention to pain relief, hydration, nutrition, hemodynamic support, supplemental oxygen, or other respiratory support, and treatment of bacterial superinfections of skin lesions should be considered when indicated to avoid bacterial overinfection of skin lesions. Skin lesions should be kept clean and dry. Pruritus should be treated with oral antihistamines or topical vaseline. In oral lesions should associate antihistamines, steroids, lidocaine and antimicrobials or anesthetic gels. Genital lesions are treated with topical anesthetics, acetatophaside, NSAID, neuropathic pain agents, or even opiate medications in severe cases. Proctitis is treated similarly with the addition of laxatives and seat baths. Headaches are treated symptomatically with hydration, acetatopine, IAES or other common acute headache treatments as appropriate [5].

Management of eye infections/complications, specifically resulting in corneal scarring and/or vision loss. Potential approaches to consider in this situation include the early involvement of ophthalmology specialists, application of lubricants, topical antibiotics and possibly topical antivirals such as trifluridine [6].

To date, there are no treatments approved by the U.S. Food and Drug Administration (FDA) specifically for monkeypox. However, in severe cases there are antiviral agents that have activity against MPXV, including cidofovir, brincidofovir (a prodrug conjugated with cidofovir lipids) and tecovirimat (TPOXX) – inhibitor of the enzyme wrapping envelope VP37 viral orthopox, treatment approved for viral infection by smallpox. In addition to antiviral agents, vaccinia intravenous immunoglobulin (VIGIV) has previously been approved by the FDA for the treatment of complications arising from vaccinia vaccination, such as progressive vaccinia and severe generalized vaccinia.

Currently, tecovirimat, cidofovir and VIGIV are available in the National Strategic Stock under Protocols of New Expanded Access InvestigationAl Drugs (EA-IND) maintained by the Centers for Disease Control and Prevention (CDC) for the treatment of OPXV infections in an outbreak scenario [6].

OPXV infection may confer cross-immunological protection among viruses of the same genus. There are no vaccines specifically designed to protect against infection and smallpox disease in monkeys. Vaccines considered for use (Vaccinia virus-based vaccines) to prevent MPXV were developed for smallpox. In a study conducted in the DRC in the late 1980s, unvaccinated home contacts of individuals with MPXV had a secondary attack rate of 9.28% compared to 1.31% for vaccinated contacts. This yielded an approximate estimate of 85% protection conferred by previous smallpox vaccination against smallpox from monkeys [6].

Complications

Monkeypox virus infection is a disease with a self-limiting course and its complications have been more frequently observed in unvaccinated individuals (74%) than in vaccinated individuals (39.5%) [12,17,18], and may be more severe in HIV-infected patients [7,19].

Currently, the most serious complications are eye infections,

which result in corneal re-epithelialization with healing epithelium and permanent loss of vision [11]. However, two fatal cases have been reported, in presumably immunocompetent men, in which the MPV infection evolved in a neuroinvasive way, coursing with encephalitis [5]. In addition, severe dehydration can occur from gastrointestinal symptoms and oral mucosal lesions [12,20].

Furthermore, it was observed that the extensive area affected by mucocutaneous lesions is associated with a higher risk of bacterial superinfection [11] and potential evolution to an exacerbated immune response, coursing with sepsis. There have been reports of cases of bronchopneumonia and respiratory failure [16,17], however these complications were more commonly observed in individuals co-infected with the Influenza virus [11].

Prognosis

Studies show that Monkeypox infection is usually self-limiting and has a low mortality rate [15], the WHO recorded 12 deaths by the end of August 2022 [20]. A 2022 systematic review performed by a Peruvian group combined 19 articles, totaling 1958 patients, recorded a death rate of 4% [20].

Most studies have not observed fatal outcomes [15], however there are reports of permanent sequelae, including skin scarring and blindness from corneal lesions [12,13].

Conclusion

Although anal manifestations such as those presented on this report are not frequently described in association with Monkeypox infeccion, most recent case reports have indicated the anorectal region as one of the most commonly affected by the appearing of the characteristic lesions of this desease. Adding up to the lesions, an intense acute pain scenario may present itself, increasing hospitalization potencial of infected patients. Therefore, it is highly important to prevent the spread of this disease, which incidence has grown significantly worldwide in the last few months.

Research Ethics Committee Approval

We declare that the patient approved the study by signing an informed consent form and the study followed the ethical guidelines established by the Declaration of Helsinki.

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