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Case Report

Zoonotic orthopox virus infection

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Abstract

Introduction:

Cowpox virus (CPXV) is a rare zoonotic orthopoxvirus infection, most commonly transmitted to humans via infected domestic cats. It typically causes necrotic skin lesions accompanied by systemic symptoms such as fever and lymphadenopathy. Diagnosis is confirmed by polymerase chain reaction (PCR) and sequencing, and management is mainly supportive.

Case Presentation:

A 64-year-old farmer presented with painful, clustered necrotic lesions on the face and neck evolving over ten days, associated with fever and lymphadenopathy. Due to the facial distribution and severity, broad empirical treatment with intravenous acyclovir and ceftriaxone was initiated. Diagnostic testing for common infectious causes was negative, and bacterial cultures showed no growth. Based on lesion morphology and relevant animal exposure, orthopoxvirus infection was suspected. PCR of skin swab material confirmed orthopoxvirus DNA, and sequencing identified cowpox/catpox virus (CPXV).

Conclusion:

This case highlights CPXV infection in an older host with prominent facial clustering, initially requiring broad empirical therapy. CPXV should be considered in patients with necrotic facial lesions and animal exposure. PCR-based diagnostics are essential for confirmation, and the clinical course is usually benign with supportive management, although residual scarring may occur.

Introduction

Cowpox virus (CPXV), genus Orthopoxvirus, are caused by Poxvirus bovis, a double-stranded DNA virus measuring 200×300 nm. The most common source of human infection is the domestic cat (as the vector), with the reservoir being rodents; however, it should also be noted that pet rats have been reported as vectors of human infection [1]**.** Human-to-human transmission of cowpox virus is considered extremely rare. Despite numerous reported cases of infections in cats, and thus a high likelihood of human exposure, relatively few human infections occur. In cats, the infection initially presents as a bite-like lesion on the front paws or head. Secondary lesions appear roughly 10 days later in terms of erythematous macules that progress to papules and eventually crusted ulcerations [2]. The typical clinical course in humans begins with erythematosus maculae. Within 7–12 days, papules or vesicles form (initially serous, later hemorrhagic). Pustules (with sterile content) develop, followed by ulceration and crusting approximately 14 days later. Additionally, flu-like symptoms such as fever and malaise may accompany the skin findings. Typical disease duration is approximately 6 to 8 weeks.

Case Report

A 64-year-old man presented with painful facial lesions that began on his neck and spread to his face over the course of ten days. He also reported fever, swollen lymph nodes and discomfort. The patient is a farmer with regular contact to cows, cats and horses. None of the animals had cutaneous lesions or illnesses, as no signs of disease were observed or reported despite regular contact. Physical examination revealed multiple erythematous, centrally depressed papules and nodules up to 1 cm in size, with hemorrhagic crusts and central necrosis, on the face and neck, as shown in Figure 1 A-C. No mucosal lesions were noted. Laboratory analysis revealed elevated C-reactive protein level (79,2 mg/l; normal range, <5 mg/dl) with a normal total leukocyte count. The patient showed no clinical or laboratory signs of immunodeficiency. He was initially started on intravenous acyclovir and ceftriaxone, along with local antiseptic treatment. Empirical treatment with acyclovir was initiated because of the acute onset of painful, necrotic, and clustered facial lesions accompanied by fever and lymphadenopathy, raising initial concern for herpesvirus infection, particularly herpes zoster or severe herpes simplex virus infection. Given the potential risk of ocular involvement and complications, early antiviral therapy was started pending definitive diagnostic results. In the following two days, serologic test results for varicella-zoster virus, herpes simplex virus, 'human immunodeficiency virus (HIV), hepatitis A and B, *Mycoplasma pneumoniae*, and syphilis were negative. The ophthalmological consultation showed no evidence of ocular involvement. Microbiological analysis from a punch biopsy of the neck showed no bacterial growth in either aerobic or anaerobic cultures. Due to animal contact in the detailed anamnesis, we considered an infection from the pox virus family. Polymerase chain reaction of skin swab material confirmed the presence of orthopox virus DNA, subsequently identifying the serotype via sequencing as cowpox/catpox virus (CPXV). Histopathological analysis was not performed because the diagnosis was established by microbiological and molecular testing, and there was no additional clinical indication for histological examination. Supportive care and wound debridement were provided. The lesions gradually resolved with scarring over the course of 2 months as shown in Figure 1 D and E.

Discussion

The clinical suspicion of a poxvirus infection, as in our patient, arises from the characteristic pockmark (central depression) of the papules as shown in Figure 1 C [3]. PCR and subsequent sequencing are diagnostic gold standard. An electron microscopy examination of vesicle fluid, crust material, or tissue may also confirm the diagnosis, revealing virions with irregular surface filaments. Additionally, virus antigen detection from skin lesions and antibody titer determination in serum via competition ELISA or antibody neutralization tests are possible [4]. Histologically, the infection is characterized by intraepidermal vesiculation and ballooning

keratinocytes with eosinophilic inclusion bodies, corresponding to aggregated viral particles. Differential diagnoses include infections with parapoxviruses (contagious ecthyma, milker's nodules), herpes simplex virus, and bacterial infections (anthrax, actinomycosis). In our patient, parapoxvirus infection was considered less likely because of the clustered facial distribution and the presence of centrally depressed, necrotic lesions, while herpes simplex virus infection was ruled out by negative serological testing and the absence of mucosal involvement. Bacterial infections, including anthrax and actinomycosis, were excluded by negative aerobic and anaerobic cultures obtained from a punch biopsy and the lack of progressive systemic deterioration. Infections generally show benign trajectories. Initial treatment is symptomatic, including local antiseptic and anti-inflammatory measures, as well as debridement of crusts and necroses. To prevent bacterial superinfection, systemic antibiotics can be administered. Specific antiviral therapy with Cidofovir or antivaccinia gamma globulin is possible but should be reserved for refractory cases due to potentially severe side effects [5]. Suspected or confirmed orthopoxvirus infections may be subject to reporting obligations depending on local regulations, and notification of the responsible public health authorities should be considered accordingly.

Statements

Statement of Ethics

Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images. Ethical approval is not required for this study in accordance with local or national guidelines. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

Bernadett Kurz, MD: conceptualization, data curation, formal analysis, investigation, methodology, validation, writing - original draft, writing - review and editing; Dennis Niebel, MD: writing - review and editing; Susanne Bauer, MD: writing - review and editing; Julian Kögel, MD: writing - review and editing; Mark Berneburg, MD: writing - review and editing; Sigrid Karrer, MD: writing - review and editing.

Data Availability Statement

The data that support the findings of this study are not publicly available due to their containing information that could compromise the privacy of research participants but are available from BK upon reasonable request

References

1. Wollina U, Hansel G, Schönlebe J. Cowpox virus infection from pet rat. Our Dermatol Online. 2018;9(1):31–34.
2. Silva NIO, de Oliveira JS, Kroon EG, Trindade GS, Drumond BP. Here, there, and everywhere: the wide host range and geographic distribution of zoonotic orthopoxviruses. Viruses. 2020;13(1).
3. Baxby D, Bennett M, Getty B. Human cowpox 1969-93: a review based on 54 cases. Br J Dermatol. 1994;131(5):598–607.
4. Bruneau RC, Tazi L, Rothenburg S. Cowpox viruses: a zoo full of viral diversity and lurking threats. Biomolecules. 2023;13(2):325.
5. Obermeier PE, Buder SC, Hillen U. Poxvirus infections in dermatology—the neglected, the notable, and the notorious. J Dtsch Dermatol Ges. 2024;22(1):56–93.

Figure Legends

Fig. 1 Clinical picture of the patient:
(A,B,C) The face, neck and upper body are covered with multiple erythematous, centrally depressed papules and nodules up to 1 cm in size, with serous and haemorrhagic crusts and a few necrotic areas.
(D,E) Two months later: Face and neck show faint erythematous postinflammatory macules and scarring, as well as hypopigmented macules.