19-21 GIUGNO ROMA
UNIVERSITA' CATTOLICA DEL SACRO CUORE CENTRO CONGRESSI EUROPA
RESEARCH and CARE: FROM BENCH, TO BEDSIDE, TO COMMUNITY
Promosso da SIMIT
16° CONGRESSO NAZIONALE ICAR
Italian Conference on AIDS and Antiviral Research
P 80
POSTER 4 YOU ICAR 2024

Challenging the Bounds of Co-Infection: A Case Study on Severe Monkeypox in an Untreated HIV Patient and the Role of Tecovirimat in Treatment Efficacy

T. Semenzin[1], G. Matusali[3], E. Sozio[2], P. Della Siega[2], F. Colavita[3], E. Cimini[3], V. Mazzotta[3], F. Maggi[3], C. Tascini[1,2], S. Lanini[1,2]

Affiliation: 1. Department of Medicine (DAME), University of Udine, 33100, Italy. 2. U.O. Malattie Infettive, Azienda Sanitaria Universitaria Friuli Centrale, Udine, Italy. 3. Laboratory of Virology, National Institute for Infectious Diseases Lazzaro Spallanzani IRCCS, Roma, Italy

INTRODUCTION

Mpox, formerly known as monkeypox, is a zoonotic viral infection caused by the Mpox virus, which is like the virus causing smallpox. People living with HIV (PLWH) are at increased risk for severe outcomes from mpox due to their compromised immune systems. This vulnerability is particularly significant for those with low CD4 cell counts or untreated HIV. The interplay between Mpox and HIV necessitates careful monitoring and management, as co-infection can complicate the clinical course and therapeutic strategies. Early diagnosis and tailored treatment are crucial to mitigate the impact of Mpox on this vulnerable population

CASE-STUDY

A 32-year-old MSM with a history of multiple unprotected sexual and untreated HIV infection, presented with a severe Monkeypox virus infection. He was referred to our unit by a local STI unit with a positive rectal swab for Mpox (Clade II) in January 2024. His clinical manifestations included fever, lymphadenopathy, and rash (about 25-30 lesions). Laboratory evaluations showed detectable HIV RNA, low CD4 counts, and high inflammatory markers (Table 1). Molecular diagnostics revealed the presence of M. hominis and U. urealyticum on the rectal swab, as well as HSV-1; M. hominis was also detected in urine and in the oropharyngeal swab. He tested negative for N. gonorrhea and C. trachomatis. The results for TPHA and VDRL were 1:5120 and <1:2, respectively. Several HPV lesions were identified on the perianal skin and the rectal swab was positive for genotypes 16,39,52,66. The patient experienced severe, intractable pain, which necessitated the initiation of opioid therapy. He began treatment with tecovirimat, ganciclovir, doxycycline, and antiretroviral therapy with BIC/TAF/FTC (Table 1). Imaging studies, including a CT scan, identified pseudonodular pulmonary lesions (Fig. 1B), and colonoscopy revealed severe proctitis without lesions in the sigma and colon (Fig. 1A). Although no respiratory tract lesions were found during bronchoscopy, a molecular test on the bronchoalveolar lavage fluid was positive for Mpox. He receiving tecovirimat for an extended duration of about 8 weeks, it was well tolerated with no signs of renal or liver toxicity observed. Despite the long and off-label treatment with tecovirimat Mpox persisted in all significant sites. Over the course of treatment, there was a noted progressive reduction in HIV following antiretroviral therapy and from the week 7 on going, HIV-RNA was undetectable. From approximately week 12, there were no longer any skin lesions attributable to Mpox. Microbiological examinations revealed reactivation of CMV infection with the presence of the virus in multiple sites (blood, rectal and oral swab) in the absence of organ localization (table 1). In addition to the infectious problems that were difficult to manage, there was the pain component which required an increase in antalgic therapy and, above all, in the psychiatric management of the patient. The patient remained hospitalized.

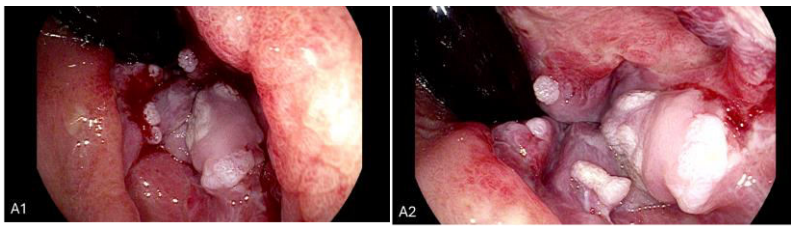
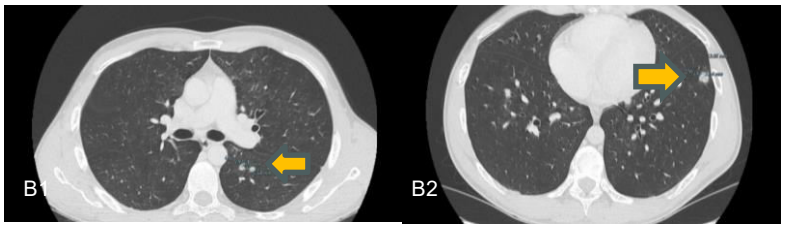


Image 1.

1 A. Severe proctitis documented by colonoscopy

1B. bilateral lung nodules documented at the chest CT scan (first week)



	Clinical marker	Time of hospital stay											
		Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7	Week 8	Week 11	Week 13	Week 16	Week 20
biochemistry	WBC (mmc)	4400	3310	1820	3180	5220	4620	7700	5200	9100	2930	6590	3530
	N/L	2.84	1.49	0.75	0.81	2.7	1.11	5.94	1.28	1,11	0,99	1,34	1,21
	CRP	20	9	8	8	5	12	17	8.5	6,5	2,6	2,1	1,1
	creatinine (mg/dL)	0.75	0.72	0.96	0.91	0.94	0.95	0.91	0.91	0,94	0,73	1,05	0,99
	AST (UI/L)	35	26	17	18	20	30	21	15	16	20	16	16
	ALT (UI/L)	39	34	19	13	18	45	24	11	11	12	11	10
HIV	CD4 (%)	101 (12%)				99 (9%)		115 (11%)		152 (8%)	208 (8%)	138 (10%)	195 (9%)
	CD4/CD8	0.2				0.1		0.1		0,1	0,1	0,2	0,1
	CMV-DNA (cp/mL) blood	492	46	< the limit				58	76	330	1280	1490	397
	HIV-RNA (cp/mL)	75900	3880			50		< the limit			< the limit		< the limit
Mpox	MPXV DNA Ct value T. lesions	20					25		On going				
	MPXV DNA Ct value T. rectal	15		26					On going				On going
	MPXV DNA Ct value T. oropharynges	21		Neg			24		On going				On going
	MPXV DNA Ct value urin	Neg					34		On going				On going
	MPXV DNA Ct value plasma	25		30			28		On going				On going
	anti-MPXV IgG	1:80		1:160			1:160						
	anti-MPXV IgM	Weak reactivity		Weak reactivity			Weak reactivity						
	anti-MPXV IgA	< 1:20		< 1:20			< 1:20						
Therapy	TARV (BIC/TAF/FTC)												
	tecovirimat												
	doxycycline												
	gancyclovir												
	TMP/SMX prophylaxis												
	valacyclovir												
	fluconazolo												

Table 1. Assessment of the temporal trend of biochemical, virological status and therapy.

WBC (White blood cells), N/L (neutrophiles and lymphocytes ratio), renal function (creatinine), flogosis index CRP (C-reactive protein), liver function (AST aspartate amino trasferare, ALT alanine aminotransferase), CD4 cell count and viral load of HIV, cytomegalovirus, DNA=cytomegalovirus DNA. cp=copy. Real-time PCR for monkeypox virus DNA was performed on skin lesions localized in different body areas, oropharyngeal swabs, rectal swabs, urine, plasma collected from day 7 of admission and still going on. Results are expressed as cycle threshold values. Serum samples collected at different times from symptom onset were tested for the presence of anti-monkeypox virus IgA, IgM, IgG, and neutralizing antibodies.

The horizontal black lines represent the various therapies used, the moment they were started, concluded, or if they are still ongoing.

CONCLUSION

This case is significant in the context of the evolving epidemiology of Mpox, following the European outbreak. The presentation underscores the importance of including Mpox in the differential diagnosis of STIs when suggestive skin lesions are present; especially in populations at risk. Furthermore, the severe manifestation of Mpox in this patient, alongside with HIV, suggests that Mpox could be considered a new AIDS-defining illness. This aligns with ongoing discussions in the medical community about the implications of labelling severe Mpox as such, considering the need for awareness against the potential for stigmatization. The well-tolerated extended use of tecovirimat without signs of toxicity adds valuable real-world evidence to the ongoing evaluation of tecovirimat's efficacy and safety profile. However, discussions regarding the actual efficacy of tecovirimat persist, emphasizing the need for continuous research.

This case highlights the critical aspects of the current understanding and management of Mpox in high-risk populations and the ongoing assessment of therapeutic options like tecovirimat. It underscores the importance of integrating clinical findings with epidemiological insights to inform public health strategies and therapeutic approaches in managing emerging infectious diseases.