

LYELL'S SYNDROME IN CAR-T TREATED PATIENTS: A CASE STUDY

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BACKGROUND AND

AIM AND OBJECTIVES

This case report explores potential triggers of Lyell's syndrome in 39-year-old woman

IMPORTANCE

Lyell's syndrome- a toxic epidermal necrolysis-is a rare and potentially lifethreatening disease that affects the skin and mucous membranes.The drugs commonly implicated in toxic epidermal necrolysis (TEN) include non-steroidal antiinflammatory drugs, chemotherapy, antibiotics and anticonvulsants. diagnosed with relapse and diffuse refractory large cell B lymphoma (DLBCL) who underwent Third Line Therapy with Axicabtageneciloleucel.After the infusion, CRS (cytokine release syndrome) was reported, which progressed from grade 1 to G2 within 3 days. This was complicated by the onset of ICANS (immune-effector cell-associated neurotoxicity syndrome) progressed to G3 within 3 days. Subsequently, the HLH/MAS framework (Hemophagocytic Lymphohistiocytosis/Macrophage Activation Syndrome) was reported. To control her persistent high fever and to reduce the risk of convulsions, was somministrated levetiracetam. Despite anti-cytokine therapies and steroids were continued, after 6 days Toxic epidermolysis affected 90% of the body surface area, confirmed by histological examination of the skin rhomboid, consistent with TEN/Lyell syndrome. Levetiracetam was discontinued.





METHODOLOGY

Medical records and National Pharmacovigilance Network were used to collect data.

RESULTS/FINDINGS

The patient was admitted to the intensive care unit for 32 days, receiving treatments comparable to those given to patients with severe burns.Drugs administered: ruxolitinib, methylprednisolone, daptomycin, amine, piperacillin/tazobactam, tocilizumab, entanercept , anakinra, and high-dose fluids. The pharmacist provided critical support to CAR-T team, playing a key role in the management of drug selection and occasionally resort to off-label use of medicines. A sterile paraffin tulle gras dressing led to re-epithelialization and disappearance of the blisters. DLBCL progression led to death 9 months later.



CONCLUSION AND RELEVANCE

The co-administration of several drugs, the lack of available data on adverse drug reactions (ADRs) in response to CAR-T, and the temporal relationship between levetiracetam and onset of ADR lead to the conclusion that a metabolite of anticonvulsivants, identified in the literature as a potential trigger, was responsible for the ADR. The decision to use anti-TNF-alpha was critical in the management of

the syndrome. A comparable ADR was subsequently reported in Eudravigilance, raising uncertainty about the potential involvement of levetiracetam as a trigger of the ADR.

No conflict of interest

