Monoclonal antibodies in covid-19 treatment: Efficacy and limitations.

Ane Sumbayev*

Medway School of Pharmacy, Universities of Kent and Greenwich, UK

Introduction

The COVID-19 pandemic, caused by SARS-CoV-2, prompted unprecedented global scientific collaboration to identify effective treatments. Among the most promising therapies are monoclonal antibodies (mAbs)—lab-engineered molecules designed to mimic the immune system's ability to neutralize viruses. These antibodies target specific components of the SARS-CoV-2 virus, particularly the spike protein, to prevent viral entry into human cells. While mAbs have shown therapeutic potential, their efficacy and limitations have also sparked debate, especially as new viral variants emerge [1].

Monoclonal antibodies for COVID-19 primarily target the receptor-binding domain (RBD) of the spike protein of SARS-CoV-2, which binds to the angiotensin-converting enzyme 2 (ACE2) receptor on host cells. By binding to this domain, mAbs block viral entry and help mark the virus for immune destruction. Most mAbs used for COVID-19 are either neutralizing antibodies or combinations (cocktails) designed to reduce the risk of resistance through viral mutation [2].

Several monoclonal antibodies received Emergency Use Authorization (EUA) from the U.S. FDA and other regulatory bodies, including: These mAbs were used to treat mild-to-moderate COVID-19 in high-risk individuals, especially those not yet hospitalized or needing supplemental oxygen [3].

Clinical trials and real-world data have demonstrated the efficacy of monoclonal antibodies in reducing COVID-19 progression, hospitalization, and mortality: The BLAZE-1 trial showed that bamlanivimab reduced viral load and COVID-19-related hospitalizations in high-risk patients [4].

Casirivimab and imdevimab reduced hospitalization or death by 70% in non-hospitalized patients with COVID-19. Sotrovimab demonstrated a 79% reduction in hospitalization or death when administered early [5].

Importantly, these treatments were most effective when given early—within 7–10 days of symptom onset. One of the major limitations of mAbs is their reduced efficacy against emerging SARS-CoV-2 variants. For instance, the Omicron variant has shown mutations in the spike protein that reduce binding affinity for several mAbs. As a result, many earlier mAbs lost efficacy and were withdrawn or restricted from use [6].

Combining mAbs targeting different spike protein epitopes helps reduce the chance of viral escape. This strategy has proven more resilient against mutations and prolongs therapeutic usefulness. For instance, casirivimab/imdevimab and bamlanivimab/etesevimab combinations have been more effective than monotherapy. Monoclonal antibody therapies are expensive to develop, produce, and administer. Their cold storage requirements and need for intravenous or subcutaneous administration pose logistical challenges, especially in resource-limited settings [7].

To be effective, mAbs must be given early in the course of infection. Once the disease progresses to severe or critical stages, their efficacy diminishes, as host-driven inflammation becomes the dominant factor in disease progression rather than viral replication [8].

Continued monitoring of variant evolution and rapid adaptation of antibody design will be essential in keeping mAbs effective. Integration with antiviral pills and vaccines may also provide a more comprehensive approach to COVID-19 management. Due to production and cost barriers, global access to mAb therapy is uneven, favoring high-income countries. Low- and middle-income nations face substantial hurdles in integrating mAbs into their COVID-19 treatment protocols [9].

To combat new variants, researchers are now developing broadly neutralizing antibodies targeting conserved viral regions, less likely to mutate. Additionally, bispecific antibodies and engineered Fc regions are being explored to improve half-life and immune function. Some mAbs, like tixagevimab/cilgavimab (Evusheld), have been approved for pre-exposure prophylaxis in immunocompromised individuals who may not respond well to vaccines. Early studies showed a significant reduction in symptomatic COVID-19 among this population [10].

Conclusion

Monoclonal antibodies have emerged as a powerful tool in the fight against COVID-19, particularly for vulnerable and high-risk populations. While they offer significant benefits in reducing severe outcomes when administered early, their efficacy is challenged by viral mutations, high costs, and logistical hurdles. Ongoing research and innovation are needed to develop more resilient and accessible antibody therapies that can adapt to the evolving pandemic landscape.

References

1. Ribas A, Wolchok JD. Cancer immunotherapy using checkpoint blockade. Sci. 2018;359(6382):1350-5.

***Correspondence to:** Ane Sumbayev, Medway School of Pharmacy, Universities of Kent and Greenwich, UK. E-mail: ane.Sumbayev@kent.ac.uk

***Received:** 03-Apr-2025, Manuscript No. AAJCIT-25-163911; **Editor assigned:** 04-Apr-2025, Pre QC No. AAJCIT-25-163911(PQ); **Reviewed:** 17-Apr-2025, QC No AAJCIT-25-163911; **Revised:** 21-Apr-2025, Manuscript No. AAJCIT-25-163911(R); **Published:** 28-Apr-2025, DOI:10.35841/aajcit-8.2.256*

***Citation:** Sumbayev A. Monoclonal antibodies in covid-19 treatment: Efficacy and limitations. J Cancer Immunol Ther. 2025;8(2):256*

2. Gubin MM, Zhang X, Schuster Het al., Checkpoint blockade cancer immunotherapy targets tumour-specific mutant antigens. Nat. 2014;515(7528):577-81.

3. Ramsay AG. Immune checkpoint blockade immunotherapy to activate anti-tumour T-cell immunity. Br J Haematol. 2013;162(3):313-25.

4. Huang AC, Zappasodi R. A decade of checkpoint blockade immunotherapy in melanoma: understanding the molecular basis for immune sensitivity and resistance. Nat Immunol. 2022;23(5):660-70.

5. Meng X, Huang Z, Teng F, Xing L, et al.,. Predictive biomarkers in PD-1/PD-L1 checkpoint blockade immunotherapy. Cancer Treat Rev. 2015;41(10):868-76.

6. Xiao Y, Freeman GJ. The microsatellite instable subset of colorectal cancer is a particularly good candidate for checkpoint blockade immunotherapy. Cancer Discov. 2015;5(1):16-8.

7. Łuksza M, Riaz N, Makarov V, et al., A neoantigen fitness model predicts tumour response to checkpoint blockade immunotherapy. Nat. 2017;551(7681):517-20.

8. Pardoll DM. The blockade of immune checkpoints in cancer immunotherapy. Nat Rev Cancer. 2012;12(4):252-64.

9. Teng F, Kong L, Meng X, et al., Radiotherapy combined with immune checkpoint blockade immunotherapy: achievements and challenges. Cancer Lett. 2015;365(1):23-9.

10. Johanns TM, Miller CA, Dorward IG, et al., Immunogenomics of hypermutated glioblastoma: a patient with germline POLE deficiency treated with checkpoint blockade immunotherapy. Cancer Discov. 2016;6(11):1230-6.

Citation: *Sumbayev A. Monoclonal antibodies in covid-19 treatment: Efficacy and limitations. J Cancer Immunol Ther. 2025;8(2):256*