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RESEARCH ARTICLE

Immunological Aspects of Rare Inflammatory Cardiomyopathies and Therapeutic Considerations

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Abstract: Rare inflammatory cardiomyopathies—including giant-cell myocarditis, eosinophilic myocarditis, cardiac sarcoidosis, and immune checkpoint inhibitor–associated myocarditis—are characterized by distinct immune-mediated mechanisms that drive myocardial injury and remodeling. Giant-cell myocarditis reflects fulminant T-cell–mediated autoimmunity; eosinophilic myocarditis is driven by eosinophil activation and cytotoxic granule release; cardiac sarcoidosis features granulomatous inflammation with dysregulated macrophage–T-cell interactions; and checkpoint inhibitor myocarditis results from unleashed autoreactive T cells. Accurate diagnosis requires integration of multimodality imaging, serology, and often endomyocardial biopsy, since immunopathology directly informs therapy. High-dose corticosteroids are the backbone of treatment across entities, but disease-specific strategies are essential: combination immunosuppression for giant-cell myocarditis, anti–IL-5 biologics as steroid-sparing agents in eosinophilic disease, methotrexate or anti–TNF-α agents in refractory cardiac sarcoidosis, and abatacept or other targeted immunomodulators in checkpoint inhibitor myocarditis. Optimal management requires balancing rapid suppression of myocardial inflammation with standard guideline-directed heart failure and arrhythmia care, alongside careful infection risk mitigation. Evidence remains largely observational, but emerging targeted therapies and imaging-guided treatment algorithms are reshaping the therapeutic landscape. Early recognition, multidisciplinary management, and tailored immunomodulation are critical to improving outcomes in these life-threatening but treatable conditions.

Keywords: Myocarditis, Immunosuppression, Inflammation, Diagnosis, Immunomodulators.

INTRODUCTION

Rare inflammatory cardiomyopathies represent a heterogeneous group of myocardial disorders in which immune-mediated inflammation, rather than ischemia or purely genetic abnormalities, is the primary driver of cardiac injury. They include entities such as giant-cell myocarditis, eosinophilic myocarditis, cardiac sarcoidosis, and immune checkpoint inhibitor–associated myocarditis. Although individually uncommon, these conditions carry high morbidity and mortality due to fulminant heart failure, malignant arrhythmias, and progression to advanced cardiomyopathy or sudden cardiac death. Rare inflammatory cardiomyopathies involve complex immunological mechanisms that significantly influence disease progression, making targeted therapeutic strategies essential for effective management (Imazio, 2025). The immunological pathways underlying rare inflammatory cardiomyopathies play a critical role in disease manifestation and progression, highlighting the importance of tailored therapeutic interventions (Trachtenberg & Hare, 2017). Although Esfandiari and Ghanbari (2018) focus on structural bracing, their emphasis on analyzing complex interconnections can be conceptually related to understanding the intricate immunological pathways in rare inflammatory cardiomyopathies, which is essential for guiding therapeutic considerations. Structured and targeted training approaches, as applied in interventional pulmonology education, highlight the importance of specialized knowledge in understanding immunological mechanisms and guiding therapeutic considerations in rare inflammatory cardiomyopathies (Malhotra & Iyer, 2024). Applying precise measurement metrics, as used in evaluating denial of service attacks, underscores the importance of accurately assessing immunological responses to guide therapeutic strategies in rare inflammatory cardiomyopathies (Thooyamani et al., 2014). Comprehensive surveillance and detection strategies, similar to those employed in cloud-based intrusion detection systems, can be conceptually applied to monitor immunological changes and optimize therapeutic interventions in rare inflammatory cardiomyopathies (Prabu & Sudhakar, 2024).

Each of these cardiomyopathies is defined not only by its

histopathological features but also by distinct underlying immune mechanisms—ranging from T-cell–mediated cytotoxicity and granulomatous inflammation to eosinophil-driven tissue injury. Recognition of these immunological patterns is crucial because they directly inform both diagnosis and therapeutic strategies. Multimodality imaging, endomyocardial biopsy, and biomarker profiling increasingly help distinguish immune-mediated inflammation from infectious or non-inflammatory cardiomyopathies, thereby guiding timely and targeted interventions. Understanding the immunological mechanisms in rare inflammatory cardiomyopathies is essential for personalized therapeutic approaches, enabling clinicians to tailor management strategies to individual patient profiles (Tymińska et al., 2022). Insights into the immunological mechanisms of rare inflammatory cardiomyopathies are crucial for developing effective therapeutic strategies and guiding future clinical management (Tschöpe et al., 2021). Analyzing complex networks, as demonstrated in computer network knowledge sharing, parallels the study of intricate immunological pathways in rare inflammatory cardiomyopathies, providing insights crucial for effective therapeutic considerations (Perera, 2018). Innovative design strategies, such as those used in millimeter-wave antenna development, can conceptually inform the creation of precise monitoring systems for immunological markers, aiding therapeutic decision-making in rare inflammatory cardiomyopathies (Sadulla, 2024).

Corticosteroids remain the cornerstone of treatment across most forms of inflammatory cardiomyopathy, but optimal management requires disease-specific tailoring. Combination immunosuppression is often necessary in giant-cell myocarditis, while eosinophilic myocarditis may respond to anti–IL-5 biologics. Cardiac sarcoidosis frequently demands long-term immunomodulation with steroid-sparing agents, and immune checkpoint inhibitor–associated myocarditis may require rapid escalation to novel agents such as abatacept in steroid-refractory cases. Importantly, immunotherapy must be integrated with guideline-directed heart failure treatment, arrhythmia management, and, in severe cases, advanced therapies including mechanical circulatory support and transplantation. Wearable sensors and IoT-based health monitoring systems offer promising tools for tracking immunological responses and guiding therapeutic interventions in patients with rare inflammatory cardiomyopathies (Sathish Kumar, 2023). Energy-efficient machine learning algorithms on embedded systems can be leveraged to analyze complex immunological data, supporting personalized therapeutic strategies for rare inflammatory cardiomyopathies (Kavitha, 2024). Emerging memory technologies, as explored in modern electronics, offer conceptual parallels for developing advanced data storage and analysis systems to track immunological markers and optimize therapeutic strategies in rare inflammatory cardiomyopathies (Usikalu et al., 2025).

Immunological Aspects of Rare Inflammatory Cardiomyopathies

Autoimmunity plays a central role in the initiation and progression of rare inflammatory cardiomyopathies. In giant-cell myocarditis, autoreactive T lymphocytes attack cardiomyocytes, resulting in myocyte necrosis and the formation of multinucleated giant cells. Eosinophilic myocarditis often arises from drug hypersensitivity or systemic eosinophilic syndromes, where dysregulated immune tolerance promotes eosinophil-mediated cytotoxicity. Cardiac sarcoidosis reflects an exaggerated cell-mediated immune response leading to granulomatous infiltration, fibrosis, and conduction system disruption. Immune checkpoint inhibitor–associated myocarditis exemplifies iatrogenic autoimmunity, where loss of peripheral immune tolerance unleashes autoreactive T cells against cardiac antigens. These mechanisms underscore the immune system's dual role as both protector and driver of m The inflammatory milieu in these cardiomyopathies is orchestrated by a network of cytokines and immune effectors. In giant-cell myocarditis, Th1 cytokines such as interferon-γ and tumor necrosis factor-α amplify cytotoxic T-cell activity. Eosinophilic myocarditis is mediated by interleukin-5, interleukin-4, and eotaxins, which promote eosinophil recruitment, activation, and survival. Sarcoid granuloma formation depends on dysregulated macrophage–T-cell interactions, with interleukin-2, interferon-γ, and tumor necrosis factor-α sustaining granulomatous inflammation. In checkpoint inhibitor myocarditis, enhanced T-cell activation results in increased release of proinflammatory cytokines, including interleukin-6, which may contribute to myocardial necrosis and systemic hyperinflammation. Clinically, elevated serum troponin, natriuretic peptides, and sometimes inflammatory markers (C-reactive protein, soluble IL-2 receptor) serve as correlates of ongoing immune-mediated myocardial damage. The interplay of immunological mechanisms in rare inflammatory cardiomyopathies informs therapeutic considerations, particularly in the context of cardiovascular involvement seen in rheumatic immune-mediated inflammatory diseases (Buch et al., 2024). Analyzing the multiple factors that influence organizational outcomes, as discussed by Zadeh and Ghahremani (2019), parallels the need to understand diverse immunological mechanisms in rare inflammatory cardiomyopathies to guide effective therapeutic considerations.

Although rare inflammatory cardiomyopathies are largely sporadic, genetic predisposition modulates immune responses. Specific HLA genotypes (e.g., HLA-DR and HLA-DQ alleles) have been associated with susceptibility to giant-cell myocarditis and sarcoidosis, suggesting that antigen presentation pathways influence disease onset. Variants in cytokine-regulating genes, such as polymorphisms in TNF-α or IL-5 pathways, may alter the intensity of inflammatory responses and shape the clinical course of eosinophilic myocarditis.

Moreover, host genetic background may determine vulnerability to immune-related adverse events from checkpoint inhibitors, though definitive markers remain under investigation. A deeper understanding of genetic drivers may facilitate precision immunotherapy, risk stratification, and earlier diagnosis in predisposed individuals. The challenges highlighted in digital education, such as adapting strategies to complex and evolving systems, can be conceptually related to addressing the immunological intricacies of rare inflammatory cardiomyopathies and informing effective therapeutic considerations (Arvinth, 2024). Precise measurement techniques, such as those exemplified by the "Baramati Quotient" in enzyme kinetics, underscore the importance of accurate assessment of immunological pathways in rare inflammatory cardiomyopathies to inform targeted therapeutic considerations (Khyade et al., 2017).

DIAGNOSTIC APPROACHES

Cardiac imaging provides noninvasive insights into myocardial inflammation and structural consequences. Cardiac magnetic resonance (CMR) is central, as it detects myocardial edema, hyperemia, and fibrosis through T2-weighted imaging, T1 mapping, and late gadolinium enhancement (LGE). Characteristic LGE patterns—such as patchy, mid-wall, or subepicardial enhancement—help distinguish inflammatory from ischemic injury. Positron emission tomography with 18F-fluorodeoxyglucose (FDG-PET) is particularly valuable in cardiac sarcoidosis, identifying areas of active inflammation and guiding immunosuppressive therapy. Echocardiography remains useful for assessing ventricular function, wall motion abnormalities, and pericardial effusion, though it lacks specificity for inflammatory etiologies. Together, multimodality imaging improves sensitivity and allows monitoring of treatment response.

Biochemical markers complement imaging in identifying active myocardial inflammation. Cardiac troponins and B-type natriuretic peptides reflect ongoing myocyte injury and wall stress, respectively, and are elevated in many inflammatory cardiomyopathies. In systemic eosinophilic syndromes, peripheral eosinophilia and elevated serum IL-5 may signal eosinophilic myocarditis. In sarcoidosis, elevated angiotensin-converting enzyme (ACE) levels, soluble IL-2 receptor, and inflammatory markers (CRP, ESR) can support systemic disease assessment. In immune checkpoint inhibitor–associated myocarditis, troponin elevation is often disproportionate to the degree of heart failure, serving as a red flag. Despite their utility, biomarkers are nonspecific, and their interpretation must be integrated with clinical and imaging data.

Endomyocardial biopsy (EMB) remains the gold standard for diagnosing rare inflammatory cardiomyopathies when histological confirmation will alter management. EMB is particularly critical for giant-cell myocarditis and eosinophilic myocarditis, where early initiation of aggressive or targeted immunosuppression can be lifesaving. Histopathology distinguishes between T-cell–mediated necrosis, eosinophilic infiltration, or granulomatous inflammation, thereby guiding tailored therapy. Molecular testing of biopsy tissue may also exclude viral persistence, important before initiating immunosuppression. Although EMB carries procedural risks and may miss patchy lesions, its diagnostic yield improves when guided by CMR or PET imaging. Current guidelines recommend EMB in fulminant or unexplained new-onset heart failure, especially when an immune-mediated etiology is suspected.

Therapeutic Considerations

Immunosuppression is the cornerstone of treatment for rare inflammatory cardiomyopathies. High-dose corticosteroids are typically the first-line therapy across most entities, rapidly reducing myocardial inflammation and improving hemodynamics. In giant-cell myocarditis, combination therapy—including corticosteroids with calcineurin inhibitors (cyclosporine, tacrolimus) and an antiproliferative agent (azathioprine, mycophenolate)—has demonstrated improved survival and delayed progression to transplant. Eosinophilic myocarditis responds swiftly to corticosteroids, often leading to rapid clinical and laboratory improvement. In cardiac sarcoidosis, corticosteroids reduce granulomatous inflammation, prevent arrhythmias, and preserve ventricular function, though long-term therapy may require gradual tapering and monitoring. For immune checkpoint inhibitor–associated myocarditis, prompt initiation of high-dose corticosteroids is critical, as early treatment correlates with improved survival.

Targeted immunomodulatory therapies address the underlying immune mechanisms and can be used as steroid-sparing agents or in refractory disease. In eosinophilic myocarditis, anti–IL-5 agents such as mepolizumab or benralizumab reduce eosinophil activity and allow tapering of corticosteroids. Cardiac sarcoidosis may respond to methotrexate, azathioprine, mycophenolate, or TNF-α inhibitors (e.g., infliximab) in refractory cases. In steroid-refractory checkpoint inhibitor myocarditis, agents like abatacept (CTLA-4 Ig) or anti–IL-6 therapy (tocilizumab) have shown promising results in case series, addressing uncontrolled T-cell–mediated injury. Selection of targeted therapy should be guided by the dominant immune pathway, disease severity, and existing comorbidities.

While immunosuppression addresses the primary immune-mediated injury, comprehensive supportive care is essential for optimal outcomes. Guideline-directed management of heart failure—including ACE inhibitors/ARBs, beta-blockers, mineralocorticoid receptor antagonists, and diuretics—helps prevent remodelling and maintain hemodynamic stability. Arrhythmia management may require antiarrhythmics,

device therapy (ICD, CRT), or ablation. Lifestyle interventions, such as sodium restriction, fluid management, moderate physical activity as tolerated, and avoidance of cardiotoxic exposures, complement pharmacologic therapy. In severe or refractory cases, mechanical circulatory support or heart transplantation may be necessary. Multidisciplinary coordination between cardiology, immunology, and transplant teams is recommended to optimize both immunologic and cardiovascular outcomes.

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RESULT

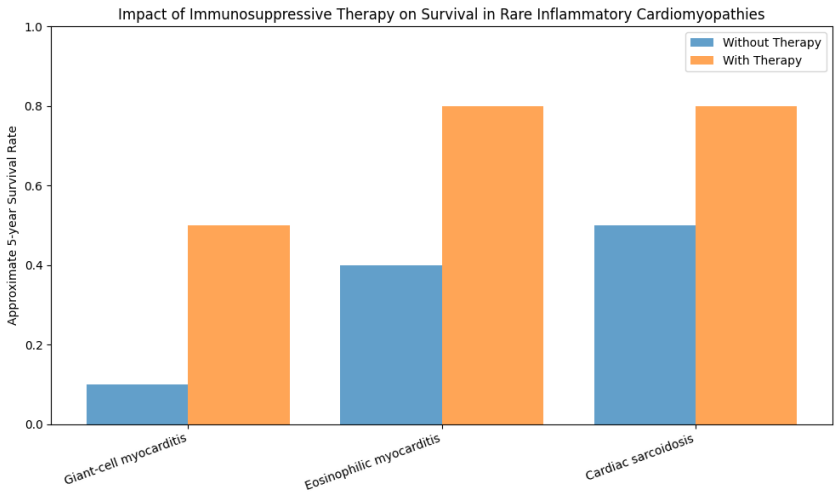

Figure1. impact of immunosuppressive therapy on survival in rare inflammatory cardiomyopathies

The figure 1 is compares 5-year survival in three rare inflammatory cardiomyopathies: giant-cell myocarditis, eosinophilic myocarditis, and cardiac sarcoidosis. The vertical axis shows the proportion of patients surviving (0–100%), while the horizontal axis lists the three conditions. Each disease has two bars: one showing survival without immunosuppressive therapy, and the other showing survival with treatment.

In giant-cell myocarditis, survival without treatment is extremely poor, around 10%. With combination immunosuppressive therapy, survival improves to roughly 50%. This highlights how aggressive the disease is, but also that early and intensive therapy can extend life, even though the overall prognosis remains guarded.

In eosinophilic myocarditis, the difference is more striking. Without treatment, survival is about 40%, whereas with corticosteroids and, in some cases, targeted therapies such as anti-IL5 agents, survival rises to approximately 80%. This underlines that this subtype responds particularly well to immunomodulation, making early recognition crucial.

In cardiac sarcoidosis, survival without treatment is around 50%, but immunosuppressive therapy (corticosteroids plus steroid-sparing agents) increases survival to about 80%. This demonstrates the benefit of therapy in controlling granulomatous inflammation, reducing arrhythmic risk, and improving long-term outcomes.

CONCLUSION

Rare inflammatory cardiomyopathies, including giant-cell myocarditis, eosinophilic myocarditis, cardiac sarcoidosis, and immune checkpoint inhibitor–associated myocarditis, are characterized by diverse immunopathological mechanisms that directly influence clinical presentation, disease progression, and therapeutic response. Understanding the underlying autoimmune and inflammatory pathways is essential for accurate diagnosis, risk stratification, and initiation of timely, disease-specific therapy. High-dose corticosteroids remain the backbone of treatment, while combination immunosuppression, targeted biologics, and steroid-sparing agents allow tailored approaches based on the dominant immune mechanism. Integration of immunomodulatory therapy with guideline-directed cardiac care, supportive management, and close monitoring is critical to improving patient outcomes.

Future research should focus on identifying precise immunologic biomarkers, elucidating genetic predispositions, and evaluating the efficacy of novel targeted therapies in prospective studies. Advancements in imaging, molecular diagnostics, and personalized immunotherapy hold promise for earlier detection, more effective treatment, and better long-term prognosis in these life-threatening but treatable conditions. Continued multidisciplinary collaboration will be key to translating mechanistic insights into optimized clinical care.

FUTURE DIRECTIONS AND CHALLENGES

Recent advances in immunology are expanding therapeutic options beyond conventional corticosteroids and broad immunosuppressants. Targeted biologics—such as anti–IL-5 agents for eosinophilic myocarditis, TNF-α inhibitors for refractory cardiac sarcoidosis, and abatacept or tocilizumab for immune checkpoint inhibitor–associated myocarditis—demonstrate promise in early clinical studies and case series. Precision immunotherapy aims to selectively modulate pathogenic immune pathways while minimizing systemic immunosuppression, reducing adverse effects, and improving long-term outcomes. Ongoing research also explores the utility of JAK inhibitors, B-cell–targeted therapies, and novel cell-based interventions in refractory cases.

Diagnosis remains difficult due to nonspecific symptoms, overlapping imaging findings with other cardiomyopathies, and variable biomarker expression. Endomyocardial biopsy, though definitive, carries procedural risks and may yield false negatives due to patchy involvement. Limited patient numbers and heterogeneity in disease presentation hinder large-scale clinical trials, resulting in most treatment recommendations being based on observational studies or expert consensus. Moreover, balancing immunosuppression to control inflammation while avoiding infection or malignancy remains a critical challenge.

Optimal management requires coordinated care among cardiologists, immunologists, pathologists, radiologists, and, when necessary, transplant specialists. Multidisciplinary evaluation facilitates timely diagnosis, selection of disease-specific immunotherapies, integration with guideline-directed heart failure and arrhythmia management, and continuous monitoring for complications. Collaborative approaches also support personalized therapy decisions, including the use of novel biologics or mechanical support in severe cases, improving both short- and long-term outcomes in these complex diseases.

REFERENCES

1. Imazio, M. (2025). Inflammatory cardiomyopathy: an update for clinicians. *Trends in Cardiovascular Medicine*.
2. Esfandiari, J., & Ghanbari, K. (2018). Comparative study of semiotic Behavior of ConnectiConnective Porch Bracing and Connective Septet Bracing. International Academic Journal of Science and Engineering, 5(1), 25–33. https://doi.org/10.9756/IAJSE/V5I1/1810003
3. Perera, T. D. P. (2018). Computer network analysis in knowledge sharing. International Journal of Communication and Computer Technologies, 6(2), 5-8.
4. Thooyamani, K. P., Khanaa, V., & Udayakumar, R. (2014). Efficiently measuring denial of service attacks using appropriate metrics. Middle-East Journal of Scientific Research, 20(12), 2464-2470.

5. Trachtenberg, B. H., & Hare, J. M. (2017). Inflammatory cardiomyopathic syndromes. *Circulation research*, *121*(7), 803-818.
6. Arvinth, N. (2024). Challenges of Digital Education in India. International Academic Journal of Social Sciences, 11(1), 01–04. https://doi.org/10.9756/IAJSS/V11I1/IAJSS1101
7. Sathish Kumar, T. M. (2023). Wearable sensors for flexible health monitoring and IoT. National Journal of RF Engineering and Wireless Communication, 1(1), 10-22. https://doi.org/10.31838/RFMW/01.01.02
8. Tymińska, A., Ozierański, K., Skwarek, A., Kapłon-Cieślicka, A., Baritussio, A., Grabowski, M., ... & Caforio, A. L. (2022). Personalized management of myocarditis and inflammatory cardiomyopathy in clinical practice. *Journal of Personalized Medicine*, *12*(2), 183.
9. Zadeh, L. K., & Ghahremani, M. (2019). Factors affecting organizational development (Case Study: Welfare office of East Azerbaijan province). International Academic Journal of Organizational Behavior and Human Resource Management, 6(1), 46–51. https://doi.org/10.9756/IAJOBHRM/V6I1/1910005
10. Kavitha, M. (2024). Energy-efficient algorithms for machine learning on embedded systems. Journal of Integrated VLSI, Embedded and Computing Technologies, 1(1), 16-20. https://doi.org/10.31838/JIVCT/01.01.04
11. Tschöpe, C., Ammirati, E., Bozkurt, B., Caforio, A. L., Cooper, L. T., Felix, S. B., ... & Van Linthout, S. (2021). Myocarditis and inflammatory cardiomyopathy: current evidence and future directions. *Nature reviews cardiology*, *18*(3), 169-193.
12. Khyade, V. B., Khyade, V. V., & Khyade, S. V. (2017). The ―Baramati Quotient‖ for the accuracy in calculation of Km value for Enzyme Kinetics. International Academic Journal of Innovative Research, 4(1), 30–51.
13. Usikalu, M. R., Alabi, D., & Ezeh, G. N. (2025). Exploring emerging memory technologies in modern electronics. Progress in Electronics and Communication Engineering, 2(2), 31–40. https://doi.org/10.31838/PECE/02.02.04
14. Buch, M. H., Mallat, Z., Dweck, M. R., Tarkin, J. M., O'Regan, D. P., Ferreira, V., ... & Plein, S. (2024). Current understanding and management of cardiovascular involvement in rheumatic immune-mediated inflammatory diseases. *Nature Reviews Rheumatology*, *20*(10), 614-634.
15. Malhotra, R., & Iyer, A. (2024). Developing an Effective Training System for Interventional Pulmonology Education through Digital Learning. *Global Journal of Medical Terminology Research and Informatics*, *1*(1), 1-8.
16. Sadulla, S. (2024). A comparative study of antenna design strategies for millimeter-wave wireless communication. SCCTS Journal of Embedded Systems Design and Applications, 1(1), 13-18. https://doi.org/10.31838/ESA/01.01.03
17. Prabu, K., & Sudhakar, P. (2024, January). A Comprehensive Survey: Exploring Current Trends and Challenges in Intrusion Detection and Prevention Systems in the Cloud Computing Paradigm. In 2024 2nd International Conference on Intelligent Data Communication Technologies and Internet of Things (IDCIoT) (pp. 351-358). IEEE.
18. Prasath, C. A. (2025). Comparative analysis of heat transfer enhancement in twisted tape inserts using nano-coated vs. traditional metal surfaces in turbulent flow regimes. Advances in Mechanical Engineering and Applications, 1(1), 23–31.
19. Sindhu, S. (2025). Blockchain-enabled decentralized identity and finance: Advancing women's socioeconomic empowerment in developing economies. Journal of Women, Innovation, and Technological Empowerment, 1(1), 19–24.
20. Veerappan, S. (2025). Voices in code: Amplifying women's experiences in tech through digital storytelling and interactive media frameworks. Journal of Women, Innovation, and Technological Empowerment, 1(1), 31–37.
21. Chen, Z., & Dahal, R. K. (2025). AI-driven big data framework for climate-resilient transportation infrastructure. Journal of Smart Infrastructure and Environmental Sustainability, 2(1), 38–44.
22. Batiha, B., & Salabi, L. (2023). Harnessing probiotics for antimicrobial resistance mitigation in dairy cattle: A sustainable One Health approach. National Journal of Animal Health and Sustainable Livestock, 1(1).
23. Sulyukova, & Budi, P. (2023). Sustainable feed formulations using agricultural byproducts: Balancing animal nutrition and environmental health. National Journal of Animal Health and Sustainable Livestock, 1(1).
24. Rozman, Z., & Rudevdagva, R. (2023). Evaluating community-based animal health delivery systems for sustainable livestock development in rural areas. National Journal of Animal Health and Sustainable Livestock, 1(1).
25. Bosco, R. M., & Aleem, F. M. (2023). Nano-encapsulated biostimulants for improving photosynthetic efficiency in capsicum under heat stress. National Journal of Plant Sciences and Smart Horticulture, 1(1).
26. Gouda, A., & Vana, C. (2023). AI-assisted phenotyping of leaf morphology for early disease detection in grapevines. National Journal of Plant Sciences and Smart Horticulture, 1(1).
27. Bates, M. P., & Jarhoumi, E. F. (2023). Exploiting plant growth-promoting rhizobacteria (PGPR) for enhanced nutrient uptake and yield in strawberry cultivation. National Journal of Plant Sciences and Smart Horticulture, 1(1).