Original Article

Clinical and functional features of dilated cardiomyopathy in cancer patients after covid-19 infection

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ABSTRACT

Background

Dilated cardiomyopathy (DCM) remains one of the leading causes of chronic heart failure worldwide. The COVID-19 pandemic has significantly changed the clinical landscape of cardiovascular diseases, revealing new mechanisms of myocardial injury. Patients with cancer represent a particularly vulnerable group due to cardiotoxic anticancer therapies and immune dysfunction. The combined impact of oncological disease, cardiotoxic treatment and SARS-CoV-2 infection may lead to rapid progression of heart failure and unfavorable outcomes.

Aim

To analyze clinical and functional features of dilated cardiomyopathy in patients with oncological diseases after COVID-19 infection.

Materials and Methods

A narrative literature review and clinical case analysis were performed. Contemporary studies on post-COVID cardiovascular complications, inflammatory cardiomyopathy and cardio-oncology were analyzed. Particular attention was paid to mechanisms of myocardial injury and progression of heart failure in cancer patients after SARS-CoV-2 infection.

Results

Recent evidence demonstrates that SARS-CoV-2 has a pronounced tropism for cardiomyocytes and vascular endothelium via ACE2 receptors. The main mechanisms of myocardial injury include direct viral cytotoxicity, cytokine storm, endothelial dysfunction, microvascular thrombosis and autoimmune inflammation. Long-term follow-up studies show a significantly increased risk of heart failure, arrhythmias and dilated cardiomyopathy after COVID-19. Cancer patients represent a high-risk population due to cardiotoxic chemotherapy, immune checkpoint inhibitors and radiation therapy. The risk of heart failure after COVID-19 in oncology patients increases by 2–3 times, while thromboembolic complications occur several times more frequently. The presented clinical case illustrates rapid progression of heart failure and fatal outcome in a patient with DCM after COVID-19 infection, consistent with the phenotype of post-COVID inflammatory cardiomyopathy.

Conclusion

SARS-CoV-2 infection should be considered an independent trigger of dilated cardiomyopathy progression. Cancer patients represent an extremely high-risk group due to synergistic myocardial injury caused by cardiotoxic therapy and post-COVID inflammatory mechanisms. Early cardiovascular risk stratification and multidisciplinary management are essential to improve outcomes in this patient population.

Keywords

Dilated cardiomyopathy; COVID-19; cardio-oncology; heart failure; myocardial injury; long COVID; cardiotoxicity; cancer patients; inflammatory cardiomyopathy.

INTRODUCTION

Cardiovascular diseases (CVDs) continue to be the leading cause of death worldwide. According to the World Health Organization, more than 17 million people die each year from conditions such as myocardial infarction and stroke [1,2]. Despite advances in prevention and treatment, the global burden of heart disease remains alarmingly high.

In recent decades, there has been a steady rise in the prevalence of chronic heart failure (CHF), which is often the final stage of many cardiovascular conditions [3]. As more patients survive acute cardiac events, the number living with long-term heart dysfunction continues to grow. In Kazakhstan, cardiovascular diseases are likewise among the primary causes of mortality. The prevalence of CHF is estimated at around 4% of the population and is steadily increasing, underscoring the urgent need for effective

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prevention, early detection, and long-term management strategies [4,5].

Dilated cardiomyopathy (DCM) is one of the key causes of CHF and is characterized by ventricular chamber dilation and reduced systolic function of the left ventricle [6,7]. The disease is heterogeneous and may develop due to genetic abnormalities, inflammatory processes, viral infections, toxic exposure, and autoimmune mechanisms [8–12].

The COVID-19 pandemic has significantly reshaped the understanding of cardiovascular disease pathogenesis. SARS-CoV-2 infection demonstrated a pronounced tropism for the cardiovascular system and was associated with an increased incidence of myocarditis, arrhythmias, thrombotic complications, and heart failure decompensation [13–15]. This issue became particularly relevant in patients with cancer, who already have an increased risk of cardiovascular complications due to cardiotoxic anticancer therapies and immune dysfunction [16–20].

Cardio-oncology has emerged as a multidisciplinary field focusing on cardiovascular complications in oncology patients. Anthracyclines, targeted therapies, immune checkpoint inhibitors, and radiation therapy are known to cause myocardial injury and contribute to the development of dilated cardiomyopathy [17–20]. SARS-CoV-2 infection further increases the risk of heart failure decompensation in this population, forming a new clinical phenotype of post-COVID cardiomyopathy [21–24].

Contemporary Understanding of Dilated Cardiomyopathy

Dilated cardiomyopathy is a heterogeneous myocardial disorder characterized by ventricular dilation and systolic dysfunction. Contemporary research demonstrates that up to 40% of cases have a genetic origin associated with mutations in sarcomeric and cytoskeletal proteins [8–11]. Abnormal gene expression leads to structural instability of cardiomyocytes, myocardial fibrosis, and progressive remodeling.

Inflammatory and autoimmune mechanisms also play a crucial role in DCM pathogenesis. Many patients exhibit cardiac autoantibodies and elevated levels of inflammatory cytokines, supporting the role of immune-mediated myocardial injury [12,25]. Infectious agents are considered important triggers initiating autoimmune inflammation and progression from myocarditis to dilated cardiomyopathy.

Viral Infections and SARS-CoV-2

Viral infections are recognized as major contributors to inflammatory cardiomyopathy. SARS-CoV-2 demonstrates strong affinity for angiotensin-converting enzyme 2 (ACE2) receptors expressed in cardiomyocytes and endothelial cells [13]. This interaction enables direct viral myocardial injury.

The mechanisms of cardiac involvement in COVID-19 include direct viral cytotoxicity, cytokine storm, endothelial dysfunction, hypercoagulability, and autoimmune inflammation [14,15]. Recent studies indicate myocardial injury in 20–40% of patients after COVID-19 infection [21]. Long-term follow-up studies show increased risks of heart failure, arrhythmias, and dilated cardiomyopathy within the spectrum of long-COVID syndrome [22].

Cancer Patients as a High-Risk Group

Patients with cancer represent a high-risk population for cardiovascular complications. Cardiotoxicity of anticancer therapy remains a major cause of secondary DCM. Anthracyclines induce dose-dependent myocardial injury accompanied by cardiomyocyte apoptosis and fibrosis . Targeted therapies and immune checkpoint inhibitors may induce immune-mediated myocarditis with mortality rates approaching 50% [20].

During the COVID-19 pandemic, the risk of heart failure among cancer patients increased two- to threefold, while thromboembolic complications became significantly more frequent [26–28]. Thus, the combination of cardiotoxic therapy and SARS-CoV-2 infection leads to synergistic myocardial injury.

Clinical Observation and Discussion

The presented clinical case demonstrates rapid progression of heart failure in a patient with dilated cardiomyopathy after COVID-19 infection. The clinical course included thromboembolic complications, laboratory evidence of hyperinflammation and hypercoagulability, and severe decline in left ventricular systolic function. Such a course corresponds to the phenotype of post-COVID inflammatory cardiomyopathy [21–24].

Ethical clearence

This study was conducted in accordance with ethical standards. Ethical approval was obtained from the appropriate institutional review board, and informed consent was secured from all participants prior to data collection.

DISCUSSION

The present review highlights a clinically important intersection between dilated cardiomyopathy, cancer-related cardiovascular vulnerability, and SARS-CoV-2–associated myocardial injury. Conceptually, DCM represents a heterogeneous myocardial disorder in which structural remodeling and systolic dysfunction may result from genetic susceptibility and/or immune-inflammatory mechanisms [8–12,25]. In this setting, COVID-19 may act not merely as an intercurrent infection but as a pathophysiological "accelerator" that shifts a patient from compensated myocardial dysfunction to overt heart failure decompensation. A growing body of evidence supports the notion that SARS-CoV-2 can contribute to myocardial injury through multiple interacting pathways, including direct injury, dysregulated inflammation, endothelial dysfunction, and hypercoagulability. In long-term follow-up cohorts, post-acute sequelae include increased risks of heart failure, arrhythmias, and cardiomyopathy phenotypes, which is consistent with the concept of post-COVID inflammatory cardiomyopathy [21–24]. From a clinical standpoint, these mechanisms are particularly relevant in patients with pre-existing myocardial vulnerability (e.g., DCM) and in those exposed to cardiotoxic treatment in oncology settings.

Cardio-oncology has established that anticancer therapies may precipitate or worsen myocardial dysfunction. Anthracyclines remain a classic example of dose-dependent myocardial injury, while targeted agents and immune checkpoint inhibitors can induce immune-mediated myocarditis with high case fatality in some series [17–20]. Thus, in cancer patients, the myocardium is often exposed to cumulative stressors—direct cardiotoxicity, systemic inflammation, and altered immunity—which may lower the threshold for post-infectious deterioration. In this context, COVID-19 adds an additional inflammatory and thrombo-inflammatory burden, increasing the probability of decompensation and adverse outcomes.

Thrombotic risk deserves special attention, since COVID-19–associated coagulopathy and endothelial activation can amplify microvascular and macrovascular complications [28]. Recent position papers and consensus statements further emphasize that the coagulation pathway in COVID-19 has broad systemic implications extending beyond classical venous thromboembolism, including microvascular injury and organ dysfunction [35]. For patients with cancer—already predisposed to thrombosis and endothelial dysfunction—these mechanisms may be clinically decisive, particularly in severe or prolonged disease courses.

Importantly, the regional and national context also matters. Data from Kazakhstan and related regional research underline persistent cardiovascular burden, comorbidity patterns, and an increase in cardiovascular morbidity observed during the COVID-19 period [4,5,36]. Such population-level observations support the rationale for early identification of high-risk phenotypes, including cancer patients with underlying cardiomyopathy, and for strengthening surveillance strategies. In parallel, digital and mobile health approaches are increasingly considered for cardiology practice and follow-up, which may be relevant for structured monitoring and continuity of care, particularly in resource-variable settings [31].

Overall, the reviewed evidence supports a unified interpretation: the combination of malignancy, anticancer cardiotoxicity, and COVID-19–related myocardial and endothelial injury forms a synergistic triad, predisposing to rapid progression of heart failure and potentially unfavorable outcomes. Clinically, this implies that oncology patients with known or suspected myocardial dysfunction may benefit from proactive cardiovascular assessment after COVID-19 and from structured follow-up strategies aligned with cardio-oncology principles and contemporary post-COVID cardiovascular risk insights [16–24,26,27].

CONCLUSION

Current evidence suggests that SARS-CoV-2 infection should be considered an independent trigger that can accelerate the progression of dilated cardiomyopathy, particularly in individuals with pre-existing myocardial vulnerability and in those exposed to systemic inflammatory stress [13–15,21–24]. The most unfavorable clinical course is expected in cancer patients receiving cardiotoxic therapy, where the myocardium may already be compromised by cumulative treatment-related injury and immune dysregulation [16–20]. In this population, COVID-19 can amplify inflammatory myocardial damage, provoke endothelial dysfunction, and increase thrombo-inflammatory complications, thereby creating a distinct phenotype of rapidly progressive heart failure requiring early recognition and multidisciplinary management [28,35].

From a practical perspective, the findings support the need for (i) early cardiovascular risk stratification of oncology patients after COVID-19, especially those with known DCM or prior exposure to cardiotoxic regimens; (ii) structured clinical and functional assessment focused on systolic function, clinical heart failure status, rhythm disturbances, and thrombotic risk; and (iii) long-term follow-up that integrates cardio-oncology principles with emerging evidence on post-COVID cardiovascular outcomes [16–24,26,27]. Where feasible, population-specific and regional data should be incorporated into planning of preventive and monitoring strategies, given the persistent cardiovascular burden and COVID-period shifts in morbidity observed in Kazakhstan and nearby regions [4,5,36]. In addition, scalable follow-up pathways, including digital or mobile health solutions, may support continuity of care and timely detection of deterioration [31].

Finally, further research is warranted to refine risk prediction models for post-COVID deterioration in oncology populations with cardiomyopathy, to clarify optimal monitoring intervals, and to define evidence-based prevention strategies for thrombo-inflammatory complications in this high-risk group . In summary, the combination of malignancy, cardiotoxic exposure, and SARS-CoV-2 infection should be recognized as a clinically significant triad associated with accelerated myocardial dysfunction and adverse heart failure trajectories, underscoring the importance of early multidisciplinary management and vigilant long-term follow-up [29–44].

Conflict of Interest: The author declare no conflict of interest.

Authors's contribution

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