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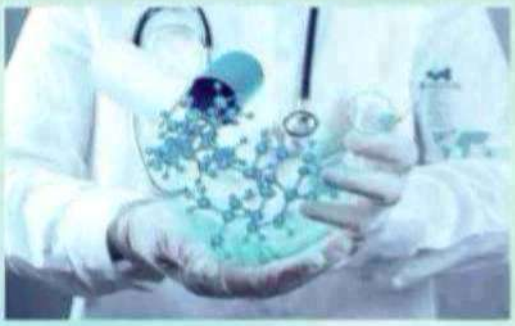

AVICENNA-MED.UZ

ISSN 2181-712X.
EiSSN 2181-2187

6 (92) 2026

ТИББИЁТДА ЯНГИ КУН
НОВЫЙ ДЕНЬ В МЕДИЦИНЕ
NEW DAY IN MEDICINE

Илмий-рефератив, маънавий-маърифий журнал
Научно-реферативный,
духовно-просветительский журнал

УЧРЕДИТЕЛИ:

БУХАРСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ ИНСТИТУТ
ООО «ТИББИЁТДА ЯНГИ КУН»

Национальный медицинский исследовательский центр хирургии имени А.В. Вишневского является генеральным научно-практическим консультантом редакции

Журнал был включен в список журнальных изданий, рецензируемых Высшей Аттестационной Комиссией Республики Узбекистан (Протокол № 201/03 от 30.12.2013 г.)

6 (92)

2026
Июнь

www.bsmi.uz
https://newdaymedicine.com
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Тел: +99890 8061882

Received: 20.05.2026, Accepted: 06.06.2026, Published: 10.06.2026

UDC 616.12-008.331.1:615.277.3

SHIELDING THE MYOCARDIUM: METABOLIC INTERVENTIONS IN THE ERA OF ANTHRACYCLINE-INDUCED CARDIOTOXICITY

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✓ ***Resume***

Anthracycline-based chemotherapy remains a cornerstone in the treatment of various hematologic and solid malignancies, but its clinical utility is fundamentally limited by dose-dependent, progressive, and often irreversible cardiotoxicity. This study reviews the molecular mechanisms of anthracycline-induced cardiotoxicity (AIC) and evaluates the therapeutic significance of metabolic shielding strategies. At the cellular level, anthracyclines induce a severe myocardial energy crisis by binding to Topoisomerase II beta, suppressing mitochondrial biogenesis, and forming iron-dependent complexes that generate massive reactive oxygen species (ROS). This dual assault paralyzes mitochondrial fatty acid β-oxidation — the heart's primary energy source. Traditional cardio-oncology strategies are primarily reactive, initiating neurohormonal blockade only after structural damage is detected via echocardiography. This paper highlights proactive metabolic interventions designed to preserve myocardial energy production without reducing antitumor efficacy. Key strategies include optimizing the L-carnitine shuttle system to restore fatty acid transport and reduce cell apoptosis, and utilizing dexrazoxane to target topoisomerase mechanisms and disrupt iron-mediated oxidative stress. The combined application of these metabolic shielding approaches improves the preservation of myocardial functional integrity and contributes to timely cardioprotective therapeutic decision-making and prevention of disease progression.

Keywords: Cardio-oncology, anthracycline cardiotoxicity, doxorubicin, mitochondrial dysfunction, L-carnitine shuttle, dexrazoxane, myocardial metabolism, reactive oxygen species (ROS).

МЕТАБОЛИЧЕСКАЯ ЗАЩИТА МИОКАРДА В ЭПОХУ АНТРАЦИКЛИН-ИНДУЦИРОВАННОЙ КАРДИОТОКСИЧНОСТИ

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✓ ***Резюме***

Химиотерапия на основе антрациклинов остается краеугольным камнем лечения различных гематологических и солидных злокачественных новообразований, однако ее клиническая эффективность ограничена дозозависимой, прогрессирующей и часто необратимой кардиотоксичностью. В данной статье рассматриваются молекулярные механизмы антрациклин-индуцированной кардиотоксичности (АИК) и оценивается терапевтическое значение стратегий метаболической защиты миокарда. На клеточном уровне антрациклины вызывают тяжелый энергетический кризис миокарда путем связывания с топоизомеразой II beta, подавления митохондриального биогенеза и формирования железозависимых комплексов, генерирующих избыточное количество

активных форм кислорода (АФК). Этот двойной удар парализует митохондриальное β-окисление жирных кислот — основной источник энергии сердца. Традиционные кардиоонкологические стратегии носят преимущественно реактивный характер, инициируя нейрогуморальную блокада только после выявления структурных повреждений при эхокардиографии. В работе освещаются проактивные метаболические вмешательства, направленные на сохранение выработки энергии в миокарде без снижения противоопухолевой эффективности. Ключевые стратегии включают оптимизацию работы карнитинового шаттла (L-карнитина) для восстановления транспорта жирных кислот и снижения апоптоза клеток, а также использование дексразоксана для воздействия на механизмы топоизомеразы и прерывания окислительного стресса. Комбинированное применение данных методов метаболической защиты повышает точность сохранения функциональной целостности миокарда и способствует своевременному принятию терапевтических решений.

Ключевые слова: Кардиоонкология, антрациклиновая кардиотоксичность, доксорубицин, митохондриальная дисфункция, L-карнитиновый шаттл, дексразоксан, метаболизм миокарда, активные формы кислорода (АФК).

ANTRATSIKLIN TA'SIRIDA YUZAGA KELADIGAN KARDIOTOKSIKLIK DAVRIDA MIOKARDNI METABOLIK HIMOYA QILISH

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✓ ***Rezyume***

Antratsiklinga asoslangan kimyoterapiya turli xil gematologik va xavfli o'smalarni davolashda asosiy poydevor bo'lib qolmoqda, biroq uning klinik samaradorligi dozaga bog'liq, progressiv va ko'pincha qaytmas kardiotoksiklik bilan cheklangan. Ushbu maqolada antratsiklin ta'sirida yuzaga keladigan kardiotoksiklik (AKK) ning molekulyar mexanizmlari ko'rib chiqiladi va miokardni metabolik himoya qilish (qalqonlash) strategiyalarining terapevtik ahamiyati baholanadi. Hujayra darajasida antratsiklinlar topoizomeraza II beta bilan bog'lanish, mitoxondrial biogenezni susaytirish va kislorodning aktiv shakllarini (KAF) ko'p miqdorda ishlab chiqaradigan temirga bog'liq komplekslarni hosil qilish orqali yurak mushagida og'ir energetik inqirozni keltirib chiqaradi. Ushbu ikki tomonlama zarba miokardning asosiy energiya manbai bo'lgan mitoxondrial yog' kislotalarining β-oksidlanishini falaj qiladi. An'anaviy kardioonkologik strategiyalar asosan reaktiv xarakterga ega bo'lib, neyrogumoral blokadani faqat exokardiografiyada strukturaviy shikastlanishlar aniqlangandan keyingina boshlaydi. Maqolada o'smaga qarshi samaradorlikni kamaytirmasdan miokardda energiya ishlab chiqarishni saqlab qolishga qaratilgan proaktiv metabolik aralashuvlar yoritilgan. Asosiy strategiyalar yog' kislotalari transportini tiklash va hujayralar apoptozini kamaytirish uchun L-karnitin shattl tizimi ishini optimallashtirishni, shuningdek, topoizomeraza mexanizmlariga ta'sir qilish va oksidlovchi stressni to'xtatish uchun deksrazoksandan foydalanishni o'z ichiga oladi. Ushbu metabolik himoya usullarining kombinatsiyalangan qo'llanilishi miokardning funksional yaxlitligini saqlash samaradorligini oshiradi va o'z vaqtida kardioprotektiv terapevtik qarorlar qabul qilishga yordam beradi.

Kalit so'zlar: Kardioonkologiya, antratsiklin kardiotoksikligi, doksorubitsin, mitoxondrial disfunksiya, L-karnitin shattli, deksrazoksan, miokard metabolizmi, kislorodning aktiv shakllari (KAF).

Relevance

The introduction of modern therapies to oncology has significantly improved overall survival times, even in metastatic cancers. The best example is that immune checkpoint inhibitors have resulted in a 5-year survival rate of approximately 50% in metastatic melanoma or renal cell carcinoma [1]. However, this trend is sometimes limited by cardiovascular side effects that may contribute to treatment discontinuation and premature death. The best example is breast cancer, where during adjuvant

treatment, discontinuation of therapy for cardiological reasons affects approximately 15% of patients and results in a significant worsening of the prognosis [2].

New cardiovascular issues or destabilization of previously well-controlled cardiovascular diseases are observed in patients treated with anticancer drugs. Therefore, in cardio-oncology it is important to properly define both permissive cardiotoxicity and toxicity requiring discontinuation of cancer therapy [3]. Patients with newly diagnosed cancer also have a high prevalence of concomitant cardiovascular disease. Available data confirmed that coronary artery disease, carotid artery disease, peripheral vascular disease, cerebrovascular disease, or heart failure could be diagnosed in 33% patients with hematologic malignancies, 43% with lung cancer, 17% with breast cancer, 26% with colon cancer, 35% with renal cell carcinoma, and 26% with head and neck cancers [4]. Cardiovascular co-morbidities can be a barrier to optimal cancer therapy. Corroborating this claim are research articles which have shown that integrators in primary care or surgery, such as PCMHs or Perioperative Surgical Homes, respectively, have a positive impact in patient care with potential reduction of costs [4, 5]. Articles such as those mentioned above show the need to expand the idea of the unattainable triangle to other specialties in medicine. One of these fields is cardio-oncology. As newer and more effective chemotherapeutic and radiotherapeutic methods have been developed in recent years, resulting in decreased mortality among cancer patients, long term cardiac-toxicities in these patients have concomitantly risen. Cancer Therapy-related Cardiovascular Toxicity (CTR-CVT) is now the leading cause of morbidity and mortality in cancer survivors [6]. It is for this reason that the field of cardio-oncology, which seeks to prevent and treat cardiac-toxicities, has sprung up as a relatively new, and rapidly evolving, specialty within medicine [6]. Given its recency as a field within medicine, larger abstract articles on cardio-oncology's overall improvement as a specialty are few. However, it is precisely because of its evolving nature that research into improving the field as a whole is important to provide guardrails to encourage the specialty to remain balanced with respect to improving quality in a cost-, and time-effective manner.

In the recent past, significant stressors have been leveraged on to the medical community, with doctors and other medical staff often experiencing the peak of this stress. These two primary stressors include the recent COVID-19 pandemic and soaring rates of inflation. Following the peak pressure of the pandemic, polls showed that that 37% of physicians perceived themselves as being overworked. These rates for other medical staff members were even higher at almost half (47.4%). What makes these statistics particularly concerning are the strong correlations found by researchers between perception of burnout and intent to leave the workforce [7]. As of July 2022, the post-pandemic healthcare workforce had lost 176,000 workers compared to pre-pandemic levels, making these findings particularly alarming [8]. By 2025, it is predicted that there will be a 50,000–80,000 person shortage in physicians [9]. Adding to this stress is the growing burden of inflation on an already burdened healthcare system. A May 2024 report by the American Hospital Association found that the economy-wide inflation rates between 2021–2023, which had risen 12.4%, were met with only a 5.2% increase in medical care reimbursements during that same time [10]. The strained financial impact that this has on the hospital is inevitably shifted on to physicians to provide more revenue for the hospital. This increased stress on the healthcare workforce provides ample justification for research into improvement on the overall healthcare industry through the lens of such concepts as the unattainable triangle. In this article, we will seek to discuss how the unattainable triangle can, and should, be applied to the field of cardio-oncology as a growing specialty in medicine, focusing on the three vertices of improved patient quality of care, reduced costs, and timely treatment.

As newer and more effective methods for cancer treatment have emerged, resulting in improved survival rates in cancer patients, the concomitant risk of long-term adverse effects has increased. Cardiovascular complications are now one of the leading causes of death in survivors of breast and colorectal cancer approximately 10 years after their diagnosis of cancer [11, 12]. However, improved outcomes have been shown through increased knowledge and monitoring of CTR-CVTs. As an example, congestive heart failure rates in patients exposed to anthracycline-based treatment have declined from estimates as high as 26% to less than 3% in the course of approximately five years with improved knowledge and interventions such as minimizing the dosing of the anthracycline drug [13]. Improvement in monitoring and treatment of cardiac conditions comes at an increased cost. While the specific costs of CTR-CVTs are relatively unknown, in 2014 it was estimated that the average cost of medical care for a male cancer survivor was $4,187 per patient per year [14]. Exacerbating these

concerns is the fact that many treatments for cardiovascular-related conditions are not covered by insurance. Examples of these services include biomarker testing during treatment, post-radiation non-invasive cardiac testing, cardiac magnetic resonance and strain imaging [15].

Additional recommendations for improving costs were discussed in a recent article from 2020 in the BMC Journal of Cardio-Oncology, which collected data on the establishment of a successful and cost-effective cardio-oncology program. Four fundamental elements were discussed in this article that highlight a comprehensive way for other institutions to develop similar programs that can subsequently decrease the overall costs of cardiological disease management in the field of oncology [15]. First is the creation of a multidisciplinary team with the integration of staff and resources from the cardiac and vascular specialties into cancer centers. This was achieved by offering cardio-oncology services at both the Cancer Center as well as the Heart and Vascular Center, which allowed for interaction of the cardio-oncologists with staff from both centers. Second was the importance of continued education for members of this evolving field. This was done by encouraging a collegial atmosphere through lectures and educational workshops for staff and trainees. The third element was engagement of the cardio-oncology physicians with professional societies such as the ACC Cardio-Oncology Advocacy Work Group and the Florida Chapter of ASCO. The fourth and final element was the development of a robust research program through data collection modalities and cooperation with other specialties (namely, oncology) and other institutions [15]. These techniques have potential for long-term positive impact in the cost of care for cardio-oncology patients.

The Root Problem: A Cellular Power Crisis

To understand why metabolic shielding is necessary, we must look at what happens inside a cardiomyocyte when it encounters an anthracycline. Unlike skeletal muscle, the human heart is a metabolic omnivore with an insatiable appetite. It generates and consumes its own weight in ATP every single day to maintain continuous mechanical contraction. Under normal physiological conditions, the heart derives roughly 60% to 90% of this energy from the oxidation of long-chain fatty acids, a process heavily reliant on healthy, high-functioning mitochondria.

Anthracyclines ruthlessly disrupt this energy-generating machinery through two primary cellular pathways:

1. The Topoisomerase II-Beta Trapping Disaster

While anthracyclines kill cancer cells by inhibiting Topoisomerase II-alpha (which is highly expressed in rapidly dividing tumor cells), they concurrently bind to Topoisomerase II-beta (Top2-beta) inside quiescent cardiomyocytes. This binding triggers a catastrophic cascade: it arrests mitochondrial biogenesis, blocks the transcription of vital respiratory chain proteins, and leaves the cell unable to repair its DNA.

2. Iron-Mediated Mitochondrial Suffocation

Anthracyclines have an exceptionally high affinity for iron. Together, they form an aggressive complex that accumulates inside the inner mitochondrial membrane, binding directly to cardiolipin. This interaction yields a massive, unmitigated surge of reactive oxygen species (ROS). The resulting oxidative stress damages the respiratory complexes, initiates lipid peroxidation, and ruptures the mitochondrial membrane, inducing cellular apoptosis (programmed cell death) and myofibrillar drop-out.

As a result, the cardiomyocyte enters an acute energy crisis. It can no longer efficiently transport or oxidize fatty acids, its primary fuel source, leaving the heart structurally weakened and metabolically starved.

Shifting the Paradigm: Metabolic Interventions

Recognizing AIC as a metabolic energy crisis opens the door to proactive, cytoprotective strategies designed to shield the heart without compromising the tumor-killing efficacy of chemotherapy.

Preserving the Fuel Lines: The L-Carnitine Shuttle System

Because anthracyclines severely impair mitochondrial fatty acid oxidation, rescuing this specific pathway is a primary target of metabolic therapy. Long-chain fatty acids cannot freely cross the inner mitochondrial membrane; they require a dedicated transport mechanism known as the carnitine shuttle.

The procedural workflow of fatty acid transport and its disruption by AIC follows five distinct phases:

- **Phase 1: Activation of Fatty Acids (Cytoplasm)** — Long-chain fatty acids are combined with Coenzyme A to form Acyl-CoA.
- **Phase 2: CPT-1 Mediated Conversion (Outer Mitochondrial Membrane)** — The enzyme Carnitine Palmitoyltransferase-1 (CPT-1) converts Acyl-CoA into Acyl-Carnitine. *Note: This step is severely inhibited by AIC-induced oxidative stress.*
- **Phase 3: Translocation (Inner Mitochondrial Membrane)** — Acyl-Carnitine is translocated across the membrane into the mitochondrial matrix via carnitine-acylcarnitine translocase.
- **Phase 4: CPT-2 Reconversion (Mitochondrial Matrix)** — Carnitine Palmitoyltransferase-2 (CPT-2) converts Acyl-Carnitine back into Acyl-CoA.
- **Phase 5: Energy Harvest (Beta-Oxidation)** — Acyl-CoA enters beta-oxidation, generating acetyl-CoA, which fuels the Krebs cycle and produces ATP.

The Therapeutic Role of L-Carnitine: By introducing exogenous L-carnitine, clinicians can help bypass the enzymatic blockades induced by oxidative stress. Supplementation supports the restoration of the intracellular carnitine pool, facilitates the transport of fatty acids into the remaining functional mitochondria, and prevents the toxic accumulation of long-chain acyl-CoAs in the cytoplasm. Furthermore, L-carnitine acts as a secondary scavenger of free radicals, stabilizing the mitochondrial membrane against ROS-mediated lipid peroxidation.

Blocking the Axis: Dexrazoxane

Dexrazoxane remains the only commercially approved cardioprotective agent specifically designed for anthracycline regimens. Its mechanism is beautifully elegant: it passes through the cell membrane and is hydrolyzed into a ring-opened structure that strongly chelates intracellular iron. By binding free iron, it prevents the formation of the highly destructive iron-doxorubicin complex, nipping mitochondrial ROS production in the bud. Additionally, dexrazoxane alters the conformation of Top2-beta, preventing the anthracycline from binding to it and preserving mitochondrial DNA transcription.

Emerging Frontiers: Exogenous Ketones and SGLT2 Inhibitors

The frontier of cardio-oncology metabolic research is exploring alternative fuel sources entirely. When the heart's ability to burn fatty acids is crippled, it naturally tries to adapt by increasing glucose utilization—a less energy-dense option.

Emerging pre-clinical models suggest that delivering exogenous ketones (such as beta-hydroxybutyrate) provides the failing, anthracycline-exposed heart with an exceptionally efficient "super-fuel" that requires less oxygen per mole of ATP generated than fatty acids. Similarly, SGLT2 inhibitors (like empagliflozin), originally designed for diabetes, are showing profound cardioprotective promise in cardio-oncology. They are hypothesized to alter systemic metabolism, shifting the heart toward ketone body utilization while reducing intracellular sodium and calcium overloads, protecting myocardial architecture from remodeling.

Conclusion: Balancing the Scales of Survival

The historical divide between oncology and cardiology is rapidly dissolving. As cancer survivorship numbers climb into the tens of millions globally, victory over a tumor cannot be celebrated if the trade-off is a severely debilitating, life-shortening cardiomyopathy.

By reframing anthracycline cardiotoxicity as a localized mitochondrial power failure rather than an unavoidable structural casualty, cardio-oncology can utilize metabolic shielding strategies—ranging from established iron chelators to optimized carnitine shuttle therapeutics and metabolic fuel switches. Ultimately, these interventions aim to ensure that a patient's heart is just as strong as the therapy that saved their life.

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Entered 20.05.2026