Cardiotoxicity

Cardiotoxicity refers to the toxic effects of substances on the heart, which can impair its structure or function. These substances can include chemicals, drugs, medications, toxins, or even radiation. Cardiotoxicity can manifest as various heart-related problems, and its severity can range from mild to severe, depending on the specific agent and the duration of exposure. Here are some key points about cardiotoxicity:

Causes: Cardiotoxicity can result from a wide range of factors, including:

Medications: Some drugs, such as certain chemotherapeutic agents (e.g., doxorubicin), antipsychotic medications, and certain antibiotics, can have cardiotoxic effects. Toxins: Exposure to certain toxins, like heavy metals (e.g., lead or mercury) or pesticides, can harm the heart. Radiation: High doses of ionizing radiation, such as in cancer treatment, can damage heart tissue. Illicit substances: The use of drugs like cocaine and methamphetamines can lead to cardiotoxicity. Chronic diseases: Conditions like hypertension, diabetes, and autoimmune diseases can indirectly lead to cardiotoxicity if left unmanaged. Manifestations: The effects of cardiotoxicity can vary widely but may include:

Arrhythmias: Irregular heart rhythms can lead to palpitations, dizziness, or fainting. Cardiomyopathy: Toxic substances can weaken the heart muscle, leading to heart failure. Myocarditis: Inflammation of the heart muscle can disrupt its normal function. Pericarditis: Inflammation of the lining around the heart can cause chest pain and discomfort. Valvular dysfunction: Some toxins can affect heart valves, impairing blood flow. Ischemia: Reduced blood flow to the heart muscle can cause angina or heart attacks. Prevention and Management: Preventing cardiotoxicity involves identifying and minimizing exposure to cardiotoxic agents whenever possible. In cases where cardiotoxicity cannot be avoided (e.g., in cancer treatment with certain chemotherapeutic agents), close monitoring and management are crucial. Management strategies may include changing medications, reducing doses, or using cardioprotective agents.

Risk Factors: Some individuals may be more susceptible to cardiotoxicity due to preexisting heart conditions, genetics, or other factors. Close monitoring and individualized care are essential for such individuals.

Early Detection: Regular medical check-ups, including cardiac assessments, can help detect cardiotoxicity early, allowing for timely intervention.

Treatment: Treatment of cardiotoxicity depends on the underlying cause and the specific manifestations. It may include discontinuing the offending agent, using medications to manage symptoms, or providing supportive care.

Research and Advancements: Ongoing research is focused on developing strategies to mitigate cardiotoxicity, such as the use of cardioprotective drugs or targeted therapies that minimize damage to the heart during cancer treatment.

It's important to note that the risk and severity of cardiotoxicity can vary widely based on the specific agent or condition involved, as well as individual factors. Anyone concerned about cardiotoxicity, especially those undergoing treatments with known cardiotoxic potential, should discuss their concerns and risk factors with their healthcare provider for appropriate monitoring and management.

Cardiotoxicity in neurosurgery

- Biomimetic extracellular vesicles derived from chimeric antigen receptor monocytes to treat glioblastoma: An efficient and safe intranasal drug delivery nanoplatform
- The CXCL1-CXCR2 Axis as a Component of Therapy Resistance, a Source of Side Effects in Cancer Treatment, and a Therapeutic Target
- Statin and Immune-Related Cardiovascular Events in Lung Cancer Patients Receiving Immune Checkpoint Inhibitors
- The citrus flavonoid nobiletin prevents the development of doxorubicin-induced heart failure by inhibiting apoptosis
- Protective Effects of Ginsenosides on Drug-induced Cardiotoxicity: A New Therapeutic Approach with Focus on Molecular Mechanisms in Cardio-oncology Field
- Cytotoxic T-lymphocyte associated protein 4 inhibitors are associated with a higher risk of cardiovascular events than programmed cell death protein 1 inhibitors in patients with melanoma
- A Bait-and-Hook Hydrogel for Net Tumor Cells to Enhance Chemotherapy and Mitigate Metastatic Dissemination
- Auto-contouring of cardiac substructures for Stereotactic arrhythmia radioablation (STAR): A STOPSTORM.eu consortium study

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Anthracyclines such as doxorubicin (DOX) are commonly used as effective chemotherapeutic drugs in anticancer therapy around the world. However, chemotherapy-induced cardiotoxicity, dilated cardiomyopathy, and congestive heart failure are seen in patients who receive DOX therapy, with the mechanisms underlying DOX-induced cardiac toxicity remaining unclear. Mitochondrial dysfunction, oxidative stress, inflammatory response, and cardiomyocytes have been shown to play crucial roles in DOX-induced cardiotoxicity. Isoliquiritigenin (ISL, 10 mg/kg) is a bioactive flavonoid compound with protective effects against inflammation, neurodegeneration, cancer, and diabetes. Here, in this study, our aim is to find out the Artemisia argyi (AA) and Ohwia caudata (OC) leaf extract combination with Isoliquiritigenin in potentiating and complementing effect against chemo drug side effect to ameliorate cardiac damage and improve the cardiac function. In this study, we showed that a combination of low (AA 300 mg/kg; OC 100 mg/kg) and high-dose(AA 600 mg/kg; OC 300 mg/kg) AA and OC water extract with ISL activated the cell survival-related AKT/PI3K signaling pathway in DOX-treated cardiac tissue leading to the upregulation of the antioxidant markers SOD, HO-1, and Keap-1 and regulated mitochondrial dysfunction through the Nrf2 signaling pathway. Moreover, the water extract of AA and OC with ISL inhibited the inflammatory response genes IL-6 and IL-1β, possibly through the NFκB/AKT/PI3K/p38α/NRLP3 signaling pathways. The water extract of AA and OC with ISL could be a potential herbal drug treatment for cardiac hypertrophy, inflammatory disease, and apoptosis, which can lead to sudden heart failure [1)]

As the intensive anti-tumor therapy and combination of multiple anti-tumor drugs, cardiotoxicity events caused by anti-tumor drugs have also increased significantly, and the incidence of cardiotoxicity also increased with survival time. Different types of anti-tumor drugs could cause all kinds of cardiotoxicity which increases the difficulties in treatment and even life-threatening. In this review, we concentrated on the targeted anti-tumor drugs such as human epidermal growth factor receptor-2 (HER2) inhibitors, tyrosine kinase inhibitors (TKIs), immune checkpoint inhibitors (ICIs), and proteasome inhibitors (PIs). The molecular mechanism of how these drugs induce cardiotoxicity is

introduced which includes several signal pathways. These drugs induced cardiotoxicity involved heart failure, hypertension, atherosis and thrombosis, QT interval prolongation, and myocarditis [2)]

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Yuan Hsieh DJ, Islam MN, Kuo WW, Shibu MA, Lai CH, Lin PY, Lin SZ, Chen MY, Huang CY. A combination of isoliquiritigenin with Artemisia argyi and Ohwia caudata water extracts attenuates oxidative stress, inflammation, and apoptosis by modulating Nrf2/Ho-1 signaling pathways in SD rats with doxorubicin-induced acute cardiotoxicity. Environ Toxicol. 2023 Sep 4. doi: 10.1002/tox.23936. Epub ahead of print. PMID: 37661764.
2)
Zhang X, Gao Y, Yang B, Ma S, Zuo W, Wei J. The mechanism and treatment of targeted anti-tumour drugs induced cardiotoxicity. Int Immunopharmacol. 2023 Apr;117:109895. doi: 10.1016/j.intimp.2023.109895. Epub 2023 Feb 18. PMID: 36806040.