Opinion

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Chemotherapeutic Medication Known As Doxorubicin Can Be Used To Treat a Variety of Tumors

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Description

Allogeneic Stem Cell Transplantation (SCT) cardiotoxicity is linked to a higher mortality rate and decreased quality of life. It is still unclear whether daunorubicin dose and cardiotoxicity following post-allogeneic Stem Cell Transplantation (SCT) are related.171 patients with Acute Myeloid Leukemia (AML) who underwent their first allogeneic SCT at our institution between the years 2005 and 2021 were the subjects of our retrospective evaluation. Cytarabine and high-dose daunorubicin were typically used as induction therapies for AML.43 patients received two courses of induction therapy with high-dose daunorubicin with a median cumulative daunorubicin dose of 310 mg/m2. A daunorubicin dose of less than 500 mg/m2 was found to be an independent risk factor for cardiotoxicity in a multivariable analysis.A decrease in LVEF was observed as the daunorubicin dose increased throughout the study; however, only a daunorubicin dose of less than 500 mg/m2 was associated with a decreased LVEF prior to SCT.

Dox Induced Significant Pathological Changes in The Cardiomyocytes

After SCT, a risk factor for cardiotoxicity is receiving high-dose daunorubicin as a second induction therapy. When selecting reinduction therapies for SCT-eligible patients with relapsed or refractory AML; this should be taken into consideration. The need to identify and treat cardiovascular conditions in cancer patients sparked the development of the cardio-oncology field. As cancer patients live longer thanks to life-saving targeted and immunologic cancer therapies that go beyond conventional chemotherapy and/or radiation therapy, the demand for this specialty continues to rise. Patients with baseline cardiovascular disease frequently require potentially cardiotoxic anticancer treatment. In addition, patients may be required to continue treatment in the event of incident cardiotoxicity associated with cancer therapy. Permissive cardiotoxicity is a novel concept that we present and discuss in this article. It is a key concept in cardio-oncology and among practicing cardio-oncology specialists. In order to achieve the best oncologic outcome while minimizing associated and possibly off-target cardiotoxicities, it places an emphasis on a proactive rather than reactive approach to the continuation of lifesaving cancer therapies. Doxorubicin primary dose-limiting effect as an anticancer agent is cardiac injury. Several intricate mechanisms, including oxidative stress, mitochondrial damage, intracellular calcium dysregulation, and apoptosis/necrosis, are associated with Dox's cardiotoxicity. Several aspects of Dox-induced cardiotoxicity are examined in this study. We looked into how Dox's acute cardiotoxicity was affected by pre-treatment with telmisartan and rosuvastatin, either in separate or combined doses. Dox induced significant pathological changes in the cardiomyocytes, according to the findings of this study. Several biomarkers of cardiac damage, including lactate dehydrogenase and cardiac troponin I, oxidative stress, such as malondialdehyde, and an inflammatory process, such as interleukin-17, experienced significant histopathological changes. We highlight the two pharmacological agents' cardioprotective effects against Dox's acute cardiotoxic effects. The significant improvement in biomarker levels and associated histological damage demonstrates this. As a clinical option for reducing the acute toxicity of Dox on cardiomyocytes, this study demonstrates the beneficial use of rosuvastatin and telmisartan either alone or in combination. Chemotherapeutic medication known as doxorubicin can be used to treat a variety of tumors. However, cardiotoxicity limits its effectiveness. Amentoflavone, a natural biflavonoid extracted from the ethyl acetate fraction of Cycas thouarsii, exhibits promising antioxidant, anti-inflammatory, and cancer-fighting properties. As a result, our research aims to discover the potential underlying mechanisms of cardioprotection and determine whether AMF could enhance cardioprotective effects against DOX cardiotoxicity. There were four categories of mice: To assess cardiotoxicity, the normal control group, the DOX group that had not been treated, and the DOX group that had been treated with AMF were given intraperitoneal injections every day for four days prior to administering doxorubicin and for an additional three days after administering DOX. AMF 80 treatment protected the group from DOX cardiotoxicity, as demonstrated by echocardiography. In addition, it effectively restored the heart weight to body weight ratio and alleviated histopathological structural changes. Serum levels of aspartate aminotransferase and Creatine Kinase-MB

(CK-MB) significantly decreased, confirming these effects biochemically.

Allogeneic Stem Cell Transplantation Cardiotoxicity Is Linked To a Higher Mortality Rate

When compared to the DOX group, AMF successfully restored the levels of expression of nuclear respiratory factor-1, mitochondrial transcription factor A, and normalized heat shock protein 27. Additionally, NADPH oxidase expression levels were significantly reduced and oxidative stress conditions were lessened by AMF. It also had significant anti-inflammatory effects by lowering immune staining of nuclear factor kabba B and suppressing IL-6 expression. In cardiac tissue, p53 immune staining and FAS ligand expression were also significantly reduced by AMF. The in vivo potential beneficial effects of AMF against acute DOX cardiotoxicity, possibly through antioxidant, anti-inflammatory, anti-apoptotic, and mitochondrial function restoration, are the subject of this first study. Perfluorooctanoic acid (PFOA) has the potential to cause developmental harm to a variety of organs, including the heart. Despite the fact that peroxisome-proliferation activated receptor alpha has been identified as PFOA's primary target, PPAR-independent effects are frequently reported. RNA-seq analysis was carried out on hatchling chicken hearts that had been exposed to vehicle or 2 mg/kg PFOA during development in order to further elucidate the mechanism of toxicity in PFOA-induced developmental cardiotoxicity. After that, RT-PCR and western blotting were used to verify the potential targets that had been found. In addition, the phenotypes resulting from the overexpression and silencing of identified target miRNAs by lentivirus in the developing chicken embryo were investigated. In chicken embryo heart developmental exposure to PFOA was found to affect 21 miRNAs and 1142 mRNAs. According to the results of RT-PCR and western blotting, miR-490-5p and its downstream targets, Synaptosome associated protein 91 and LY6/PLAUR domain containing 6, were found to be significantly affected by PFOA exposure among the identified differentially expressed miRNAs. While lentivirus silencing miR-490-5p alleviated PFOA-induced changes, lentivirus overexpressing miR-490-5p mimicked the phenotype induced by PFOA exposure. The expression of SNAP91 and LYPD6, two downstream target genes, also exhibited similar patterns. In conclusion, the fact that PFOA-induced developmental cardiotoxicity in chicken embryos is associated with miR-490-5p and its downstream genes SNAP91 and LYPD6 may aid in elucidating the underlying mechanism.