Cardiac Toxicity Related to Cancer Treatment

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Cardiac toxicity is a dose-limiting toxicity that may occur during treatment or as a late effect of therapy. Chemotherapy, radiation therapy (RT), biotherapy, and targeted therapy contribute to cardiovascular compromise that can result in cardiomyopathy, congestive heart failure (CHF), dysrhythmias, and myocardial ischemia. Advances in treatment using limited multiagent chemotherapy and reduced volume of radiation treatment to the heart contribute to minimizing late cardiac toxicity. However, cardiac toxicity remains a cause of major morbidity and mortality among childhood, adolescent, and some adult cancer survivors (Chronowski et al., 2003; Donaldson, Hancock, & Hoppe, 1999; Mertens et al., 2001).

Data from the Childhood Cancer Survivors Study indicate that childhood and adolescent cancer survivors who lived more than five years after treatment were 8.8 times more likely to die from cardiac-related events (Mertens et al., 2001). Adults who receive high-dose chemotherapy for breast cancer or lymphoma and standard treatment for non-small cell lung cancer, as well as women who receive treatment for advanced breast cancer, also may experience cardiac dysfunction following treatment. Given the growing number of long-term cancer survivors, nurses must be informed about the potential for treatment-induced cardiotoxicity. This article reviews cardiotoxic treatments, signs and symptoms of cardiotoxicity, cardioprotective measures, and clinical implications.

Cardiac toxicity is a dose-limiting toxicity that may occur during cancer treatment or several years after therapy ends. Cardiac toxicity may be caused by chemotherapy, biotherapy, and radiation therapy and may result in cardiomyopathy, congestive heart failure, dysrhythmias, and myocardial ischemia. The risk for developing cardiac toxicity varies based on type of treatment, patient age, presence of preexisting or concurrent heart disease, and concomitant treatment. Patients at high risk require careful evaluation and monitoring during and in the years following therapy to detect cardiac changes. Fortunately, cardioprotective agents and newer radiation therapy techniques decrease the risk for treatment-related cardiac toxicity. Oncology nurses can become more informed in the assessment of cardiac toxicity and can arm themselves with knowledge about early identification of symptoms as well as specific agents and treatments that increase risk for cardiac toxicity.

**Key Words:** cardiotoxin, anthracyclines, radiotherapy

**Chemotherapy-Induced Cardiac Toxicity**

Anthracyclines, alkylating agents, 5-fluorouracil (5-FU), and paclitaxel are the drugs most often associated with cardiac toxicity. These agents are associated with cardiomyopathy, CHF, dysrhythmias, and myocardial ischemia.

**Anthracyclines**

Anthracyclines are used to treat a variety of cancers and are the agents most widely associated with irreversible cardiomyopathy. Cardiotoxicity is thought to result from the release of free radicals during treatment that damage myocytes (cells of the myocardium). This damage leads to loss of myocardial contractility and eventual cell death (Hochster, Wasserheit, & Speyer, 1995; Shan, Lincoff, & Young, 1996; Speyer & Freedberg, 2000). Recent evidence indicates that oxidative stress and mitochondrial dysfunction also may be key factors in the pathogenic process (Santos, Moreno, Leino, Froberg, & Wallace, 2002).

Although acute anthracycline-induced toxicity is rare (Shan et al., 1996), chronic conditions such as cardiomyopathy and CHF may develop over time and may be severe. Thomas, Le, and Fiere (2002) described three long-term survivors of acute promyelocytic leukemia who developed anthracycline-induced CHF that required cardiac transplantation.

Doxorubicin has been the most widely studied cardiotoxic agent in adult and pediatric patients. Daunorubicin, an agent primarily used in treating acute leukemia, has a cardiotoxic risk profile similar to doxorubicin. However, epirubicin and idarubicin, analogs similar to doxorubicin, are associated with cardiac toxicity to a lesser degree than doxorubicin.
Overall, the risk of anthracycline-induced cardiac toxicity varies from 2%–23% (Steinherz, Steinherz, Tan, Heller, & Murphy 1991; Von Hoff et al., 1979). Several factors increase the risk of anthracycline-induced cardiac toxicity. These include age (i.e., more than 70 years old), pre-existing heart disease or hypertension, previous anthracycline therapy, concomitant cyclophosphamide administration, and previous history or concurrent administration of RT to the chest and mediastinum (Allen, 1992; Dunn, 1994; Hochster et al., 1995; Speyer & Freedberg, 2000; Steinherz et al.; Von Hoff et al.).

A total cumulative lifetime doxorubicin dose greater than 550 mg/m² and type of administration schedule (i.e., bolus versus continuous infusion) increase cardiac risk (Chu & DeVita, 2001; Dunn, 1994). Lower doses of doxorubicin given over longer periods of time reduce cardiotoxicity without compromising its antitumor effect (Allen, 1992).

Late cardiac toxicity may occur several years after cancer treatment. Steinherz et al. (1991) reported a 23% incidence of late cardiac abnormalities (e.g., cardiac failure) and conduction disorders (e.g., dysrhythmia) in pediatric patients who received anthracycline therapy 4–20 years earlier (Postma et al., 2002). About 5% of patients who survive 10 years after anthracycline-containing chemotherapy may experience a new onset of cardiac symptoms such as CHF or dysrhythmia (Allen, 1992). Childhood and adolescent cancer survivors also may have marginal cardiac reserves and may develop late-onset CHF at the start of vigorous exercise programs or during pregnancy (Donaldson et al., 1999).

Measures used to reduce the risk of cardiac toxicity include limiting the total lifetime dose of doxorubicin to 550 mg/m². In 1979, Von Hoff et al. suggested the use of smaller, divided doses instead of large boluses, even when the cumulative dose remains the same. Additionally, longer infusion times have been shown to reduce the risk of toxicity (Doroshow, 1991). A 75% decrease in CHF was seen when doxorubicin, at total cumulative doses less than or equal to 450 mg/m², was administered as a continuous infusion rather than a bolus infusion (Hortobagyi et al., 1989).

Mitoxantrone

Mitoxantrone is a member of the androstenedione class of synthetic antitumor agents. Its chemical structure, based on a quinone ring, is similar to that of doxorubicin (Koutinos et al., 2002). Cardiac effects such as CHF and cardiomyopathy may be less severe in comparison to doxorubicin (Chu & DeVita, 2001; Koutinos et al.). This agent needs to be used with caution in people with prior mediastinal radiation, cardiovascular disease, or prior anthracycline exposure. In anthracycline-naïve patients, toxicity risk increases when a cumulative dose of 160 mg/m² has been reached (Allen, 1992). For patients with previous anthracycline exposure, cardiotoxicity risk increases at a cumulative dose of 100 mg/m² (Allen).

Anthracycline Combinations

Taxanes in combination with anthracyclines: Several studies have examined the relationship between anthracyclines and taxanes and demonstrated that, when given together, they may increase the risk of CHF (Biganzoli et al., 2003; Perez, 2001; Sparano, 1999; Valero, Perez, & Dieras, 2001). Perez concluded that paclitaxel decreases the clearance of doxorubicin when it is given shortly after doxorubicin. Paclitaxel increases exposure to doxorubicin, thereby increasing the risk of toxicity. However, doxorubicin and paclitaxel can be administered safely when the doxorubicin dose is limited to a cumulative dose of 340–380 mg/m². High tumor response occurs without increased cardiac toxicity (Perez).

Valero et al. (2001) suggested several ways to minimize the risk of cardiotoxicity when paclitaxel is given in combination with doxorubicin. These include limiting individual dosing of doxorubicin to no more than 50 mg/m² per dose, limiting the total cumulative doxorubicin dose to 360 mg/m², and separating infusion times between paclitaxel and doxorubicin by 4–24 hours.

The increased risk of cardiac toxicity appears to be limited to paclitaxel and doxorubicin. Cardiac toxicity risk does not appear to increase when doxorubicin is combined with docetaxel. Higher doses of doxorubicin also may be given with docetaxel (Valero et al., 2001).

Trastuzumab in combination with anthracyclines: Trastuzumab is a monoclonal antibody primarily used to treat breast cancer in women who overexpress the HER-2 gene. Trastuzumab may increase the risk of cardiac dysfunction in patients with prior cardiac impairment or when given in combination with doxorubicin. Decreases in left ventricular ejection fraction (LVEF), cardiomyopathy, and CHF have been observed. Although the exact mechanism of action is unclear, anthracyclines are thought to “prime” HER-2 cells in the myocardium (Schneider, Chang, & Garratt, 2002). When trastuzumab is given, the targeted cells in the myocardium become damaged. Additionally, trastuzumab may have a direct effect on the myocardium that becomes apparent when other chemotherapy agents associated with cardiac toxicity are used (Schneider et al.).

Seidman et al. (2002) evaluated the risk of cardiac dysfunction with trastuzumab in clinical trials and found differences in risk. For example, trastuzumab increased cardiac risk 3%–7% when given alone, 13% when given with paclitaxel, and 27% when combined with anthracycline and cyclophosphamide. Multiple gated acquisition (MUGA) scans are recommended prior to treatment for patients with preexisting cardiac disease to obtain baseline cardiac function and monitor changes. Cardiac dysfunction related to trastuzumab responded well to standard therapy (Frankel, 2000).

Cyclophosphamide

Cyclophosphamide, an alkylating agent, is not associated with cardiac toxicity at standard doses but may increase the risk slightly when given in high doses, such as with blood and marrow transplant (BMT) regimens. When cyclophosphamide is given with doxorubicin, the risk of cardiac toxicity also increases. Hochster et al. (1995) reported that patients may have reversible electrocardiogram (ECG) changes with cyclophosphamide doses of 120–240 mg/kg given over one to four days.

Fatal hemorrhagic myocarditis is a very rare cardiac toxicity associated with high-dose cyclophosphamide (Hochster et al., 1995; Shanholzt, 2001). This toxicity does not appear to be cumulative but rather has an acute and rapid progression, and supportive treatment usually is rendered.

Murdych and Weisdorf (2001) reviewed the medical records of 2,821 patients who received BMT at the University of Minnesota and found that less than 1% experienced serious or fatal cardiac toxicity within 100 days of BMT. Thirteen patients experienced fatal cardiac events and another 13 experienced life-threatening cardiac events (4 with pericarditis and 9 with cardiac arrhythmias). These results suggest that with appropriate pretransplant evaluation, high-dose cyclophosphamide in the preparative phase does not result in frequent cardiac toxicity.

Chemotherapy and Biotherapy Associated With Dysrhythmias

Dysrhythmias, such as bradycardia and tachycardia, most often are seen in patients...
with preexisting cardiac conditions and may be detected at the start of treatment by a 12-lead ECG. Dysrhythmias related to chemotherapy vary in intensity and usually are asymptomatic. Generally, no treatment is recommended unless patients develop signs of cardiac compromise (Camp-Sorrell, 1999).

**Paclitaxel:** Paclitaxel is a microtubule agent used to treat ovarian, breast, and non-small cell lung cancers, as well as Kaposi’s sarcoma. Paclitaxel is associated with transient, asymptomatic bradycardia in about 30% of patients receiving a continuous infusion of the drug (Chu & DeVita, 2001; Hochster et al., 1995; Speyer & Freedberg, 2000). One case report of paclitaxel-associated tachycardia is reported in the literature (Fairve, Goldwasser, Soulie, & Misset, 1997). Cardiac symptoms can occur during initial paclitaxel infusion and may recur with subsequent infusions. Also, symptoms may worsen with concomitant cisplatin administration (Allen, 1992). Current evidence does not suggest that paclitaxel is associated with cumulative cardiac toxicity (Hochster et al.).

**Amsacrine:** Amsacrine, an alkylating agent used in treating acute myeloid leukemia, is associated with atrial arrhythmias and QT prolongation within a short time period after administration (Chu & DeVita, 2001; Speyer & Freedberg, 2000; Weiss et al., 1986). Increases in premature ventricular contractions and ventricular tachycardia also may occur (Camp-Sorrell, 1999). The exact mechanism is unknown; however, amsacrine may affect depolarization and repolarization of the heart (Weiss et al.). Hypokalemia may be a risk factor for the development of serious arrhythmias, but problems can occur even with serum potassium levels within normal limits (Weiss et al.). Less than 1% of patients who receive amsacrine develop a life-threatening arrhythmia (Allen, 1992; Speyer & Freedberg).

**Interleukin 11:** Interleukin 11 (Neumega®, Wyeth, Madison, NJ) has been associated with syncopal episodes and atrial arrhythmias (Speyer & Freedberg, 2000; Tepler et al., 1996). Cardiovascular events are associated with fluid retention and increased plasma volumes (Tepler et al.). Most often, arrhythmias resolve without clinical intervention (Rust, Wood, & Battiato, 1999; Tepler et al.). However, interleukin 11 should be used with caution in patients with a history of atrial arrhythmias or CHF, elderly patients, and those with previous anthracycline treatment or higher total doses of anthracycline therapy (Rust et al.).

### 5-Fluorouracil and Acute Coronary Symptoms

5-FU is an antimetabolite that has been associated with the development of angina and acute myocardial infarction (Anand, 1994; Hochster et al., 1995; Keefe, Rois-tacher, & Pierri, 1993; Sasson et al., 1994; Tsavaris et al., 2002). Although the incidence of cardiac toxicity is rare (1.6%–2.3%), 5-FU–associated toxicity can be significant (Anand). Cardiac symptoms have been noted with bolus and continuous infusions, with continuous infusions resulting in more frequent cardiac episodes (Tsavaris et al.). Symptoms include chest pain, nausea, and diaphoresis and are relieved once the infusion is stopped (Anand). Patients also may be treated with nitroglycerine therapy (Hochster et al.; Tsavaris et al.). Ischemia may be caused by drug metabolite-mediated increases in coronary vasomotor tone and spasms (Anand; Keefe et al.). Capecitabine, an oral prodrug of 5-FU, also was associated with reversible cardiac side effects in two case reports (Bertolini et al., 2001; Singer, 2003).

### Cardioprotective Agents

Advances in cancer care have led to the development of agents and techniques that either protect or minimize cardiac toxicity. Cardioprotective measures begin with assessment of patients prior to start of therapy, taking into consideration the current cardiac status and any history of cardiac dysfunction. Awareness of specific agents that are associated with cardiac dysfunction is useful in evaluating related symptoms.

MUGA scans are a standard evaluation tool to assess baseline and subsequent cardiac function in patients receiving anthracycline therapy. MUGA scans also have been recommended for patients receiving mitoxantrone and trastuzumab therapy. The scans measure changes in cardiac wall motion and LVEF. Baseline studies must be completed prior to initiating anthracycline therapy because agents such as doxorubicin have the potential to damage myocytes in as little as a few days to weeks following the first dose (Allen, 1992). Nousiainen, Jantunen, Vanninen, and Hartikainen (2002) found that early decline in LVEF values of more than 4% after lower cumulative doses of doxorubicin (200 mg/m²) were administered was predictive of doxorubicin toxicity in patients with lymphoma.

New agents have been developed to help reduce the risk of cardiotoxicity. For example, Doxil® (doxorubicin hydrochloride encapsulated in long-circulating Stealth® liposomes, Alza Corporation, Mountain View, CA) may have the potential to reduce the risk of cardiac toxicity (Schwonzen, Kurbacher, & Mallmann, 2000). However, long-term data about the use of cumulative doses of Doxil still are limited in determining its effect on the myocardium. Thus, the drug is assumed to have myocardial toxicity similar to conventional doxorubicin. Prior use of other anthracyclines reduces the total Doxil dose that can be given without increasing cardiac toxicity. Cardiac toxicity also may occur at lower cumulative doses in patients with prior mediastinal radiation or those who are receiving concurrent cyclophosphamide therapy. Doxil generally is recommended for patients with a history of cardiovascular disease only when the benefit outweighs the risks.

Acute infusion-associated reactions that mimic cardiac events, such as flushing, shortness of breath, facial swelling, headache, chills, back pain, tightness in the chest or throat, or hypotension, have occurred in approximately 5%–10% of patients treated with Doxil. In most patients, reactions resolve over several hours to a day once the infusion is stopped. In some patients, the reaction resolves with slowing the infusion rate. Doxil administration is recommended at an initial rate of 1 mg per minute to minimize the risk of infusion reactions (Alza Corporation, 1999).

**Dexrazoxane** (Zinecard®, Pfizer, Inc., New York, NY) is a cardioprotective agent used to prevent and minimize cardiomyopathy associated with doxorubicin. Dexrazoxane is thought to bind to free and bound iron and reduces the formation of doxorubicin-iron complexes, thus preventing free radical formation that causes cardiac damage (Chu & DeVita, 2001; Wiseman & Spencer, 1998). Several investigators (Hensley et al., 1999; Seymour, Bramwell, & Moran, 1999; Wiseman & Spencer) believed that dexrazoxane may have a role in reducing anthracycline-induced cardiac damage. However, data are limited as to the effect of dexrazoxane in tumor control and long-term survival. Thus, dexrazoxane is recommended for use in patients with metastatic disease who are sensitive to anthracycline therapy and have reached or are near the maximum-tolerated cumulative dose. Dexrazoxane is well tolerated in patients with cancer, and side effects are limited to mild myelosuppression and mild nausea and vomiting (Chu & DeVita).

After treatment ends, cardiac imaging studies such as ECG or MUGA may be helpful in

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identifying cardiac dysfunction, but current practice does not support their routine use for monitoring asymptomatic patients (Sparano, Brown, & Wolff, 2002). Other modalities, such as nuclear medicine scintigraphy and endomyocardial biopsy, as well as evaluation of circulating markers such as troponins, also may be useful in identifying patients at risk for myocardial damage; however, costs limit their routine use (Sparano et al.).

Radiation-Induced Cardiac Toxicity

RT can affect the cells of the myocardium when the heart is in or near the treatment field. RT affects cardiac tissue by altering cellular structures in the connective tissue of the myocardium. In addition, RT causes delayed damage to the surrounding capillary endothelial cells. As a result, cardiac enzyme activity is decreased, ultimately compromising the contractility and conductivity of cardiac cells. The extent of cardiac damage is related to the RT dose, volume, and treatment technique. RT-induced cardiac toxicity may be found when ECGs are performed and sometimes is discovered only when patients present emergently with cardiac-related events (Adams, Hardenbergh, Constine, & Lipshultz, 2003; Rubin, Constine, & Williams, 1998).

Pericardial disease is the most common form of radiation-induced cardiac damage, but myocardial infarction, ischemic heart disease, cardiomyopathy, and coronary artery disease also have been linked to RT (Gagliardi, Lax, & Rutqvist, 2001; Stewart, Fajardo, Gillette, & Constine, 1995). Damage to the myocardium, although rare, occurs several years after RT is completed (Applefeld et al., 1981; Stewart et al.). Several Italian researchers (Piovaccari et al., 1999; Zinzani et al., 1996) identified a 10%–11% risk for delayed cardiac complications in patients receiving irradiation to the chest for Hodgkin’s disease years after treatment had ended.

Researchers also have noted that patients with breast, lung, and esophageal cancers are at risk for cardiac toxicity if they received radiation to the chest (Martel, Sahijdak, Ten Haken, Kessler, & Turrisi, 1998; Muren, Maurstad, Hafsuland, Anker, & Dahl, 2002; Pierce et al., 2002; Stewart et al., 1995). Pericarditis has been noted in patients who have received radiation dose fractions of 3.5 Gy per day up to a total dose of 49 Gy (Martel et al.). Stewart et al. reported that when a large heart volume is in the treatment field, doses up to 40 Gy in 20 fractions over four weeks were well tolerated but higher doses were associated with an increased risk of cardiac toxicity. A small heart volume included in the treatment field with higher radiation doses up to 60 Gy in 30 fractions over six weeks also were tolerated. Doses above this level increased the risk of cardiac toxicity (Stewart et al.). When mediastinal radiation is combined with anthracyclines, cardiac toxicity may occur at lower radiation doses (Chronowski et al., 2003).

Ideally, the best plan of action is to avoid damage to the myocardium while optimizing RT to the targeted area. Today, many advances in RT now limit the amount of exposure to vital organs such as the heart and lungs. For example, traditional, rectangular fields of radiation are being replaced by three-dimensional conformal tangential irradiation (CTI) and intensity-modulated RT (IMRT). These techniques have been shown to spare normal tissue from RT-induced cardiac dysfunction (Hurkmans, Cho, Damen, Zijp, & Mijnheer, 2002; Muren et al., 2002; Pierce et al., 2002).

RT-induced cardiac toxicity can be evaluated by measuring normal tissue complication probability (NTCP) values. Hurkmans et al. (2002) studied the effects of radiation on patients with breast cancer and concluded that conformal tangential fields reduced the NTCP value to the heart by 30% when compared to standard rectangular fields. IMRT further reduced the NTCP value by 50%. These techniques target the tumor during treatment and help protect vital organs such as the heart (see the “Clinical Q&A” column on p. 587 of this issue for an in-depth discussion).

Pericardial Effusion

RT-induced pericardial effusion is a condition that occurs when fluid is unable to drain through the lymph system and becomes trapped in the pericardial space. Fluid can accumulate, restrict the heart’s ability to contract, and, eventually, cause cardiac tamponade (Fristoe, 1998). Risk factors include direct metastasis to the heart, radiation fields that include a volume of heart tissue, and concomitant chemotherapy. Symptoms of pericardial effusion may be nonspecific and usually develop slowly. Patients have reported dyspnea, nonproductive cough, chest pain, fatigue, weakness, and dizziness. Distant heart sounds, friction rubs, pericardial dullness, and cardiac enlargement also may be apparent on examination (Lawler, 1999). As symptoms worsen and cardiac tamponade develops, patients may find some relief from their symptoms by sitting up and leaning forward. A paradoxical pulse may develop, but ascertaining whether other cardiac or respiratory problems exist may be difficult (Fristoe).

Treatment for symptomatic patients includes pericardiocentesis to drain the fluid. Often, this fluid is sent for evaluation for metastasis or infective causative agents. If effusion is a recurring problem, a pericardial catheter may be placed to prevent reaccumulation of fluid. Sclerosing agents also can be instilled to help prevent fluid reaccumulation (Fristoe, 1998).

Pericarditis and Fibrosis

Pericarditis, inflammation of the lining that surrounds the heart, usually occurs within the first year after receiving RT but may occur at any time. Radiation damages the endothelial cells of the myocardium and causes edema that ruptures capillaries and small arteries. The resulting inflammation can cause pericardial fibrosis and constrictive changes that limit the filling of the heart’s chambers (Fristoe, 1998). Eventually, fluid can accumulate in the pericardial space and cause cardiac tamponade (Camp-Sorell, 1999).

Symptoms of pericarditis are similar to those of an effusion and include mild shortness of breath and severe, sharp, localized chest pain that worsens when moving, lying flat, or breathing deeply. Patients may experience some relief when they sit up and lean forward. Other symptoms include pericardial friction rub, mild peripheral edema, and decreased peripheral pulses (Camp-Sorell, 1999). ECGs may show ST-T segment changes and QRS complex voltage decreases (Fristoe, 1998). Treatment includes administration of nonsteroidal anti-inflammatory drugs and antipyretics for fever. Corticosteroids often are administered during the acute phase. Patients who are hemodynamically unstable or symptomatic may require pericardiocentesis if cardiac tamponade develops.

Clinical Implications

Cardiac toxicity is a dose-limiting toxicity of cancer therapy. New combinations of chemotherapy-limiting total cumulative doses, chemoprotective agents, and innovative radiation techniques, such as IMRT and CTI, have helped to decrease the risk of cardiac toxicity. Given the increasing number of cancer survivors, oncology nurses can help to identify patients who are at risk for developing cardiac dysfunction.
during treatment and after therapy ends, educate patients about possible signs and symptoms, and advise them on when to seek prompt attention.

Patient assessment prior to treatment focuses on evaluation of prior cardiac history and potential cardiotoxic treatment regimens that have been prescribed. Patients may need to have additional imaging scans and studies prior to treatment, and nurses are instrumental in educating and preparing patients for these studies.

Patient assessment during treatment focuses on identifying symptoms of chemotherapy- and radiation-induced cardiac changes. Angina, dysrhythmias, dyspnea at rest and with activity, hypotension, diaphoresis, venous jugular distention, peripheral edema, ECG changes, and third and fourth heart sounds need to be evaluated promptly. New complaints need further investigation. Therapy may need to be stopped until symptoms resolve or discontinued until investigated further. Patient assessment after treatment ends focuses on educating patients about long-term risk of cardiac toxicity and instructing them to inform their oncology team so they can receive timely intervention. Oncology nurses are key members of the healthcare team in assessing and monitoring patients at risk for cardiotoxicity during and following treatment.

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Cardiac Toxicity Related to Cancer Treatment

- Cardiac toxicity related to cancer treatment is a cause of morbidity and mortality among childhood, adolescent, and adult cancer survivors.

- Anthracyclines (such as doxorubicin and daunorubicin), alkylating agents (such as high-dose cyclophosphamide and amscarine), 5-fluorouracil, and paclitaxel are the chemotherapy agents most often associated with cardiac toxicity.

- A total cumulative lifetime doxorubicin dose greater than 550 mg/m² and type of administration schedule (bolus infusion) increase cardiac toxicity risk.

- Concurrent administration of anthracyclines and taxanes increase the risk of congestive heart failure.

- Dexrazoxane is a chemoprotective agent used to prevent and minimize cardiomyopathy associated with doxorubicin.

- Pericardial disease is the most common form of radiation-induced cardiac damage. Myocardial infarction, ischemic heart disease, cardiomyopathy, and coronary artery disease also have been linked to radiation therapy.

- New combinations of chemotherapy-limiting total cumulative doses, chemoprotective agents, and innovative radiation therapy techniques such as intensity-modulated radiation therapy and conformal tangential irradiation reduce the risk of treatment-induced cardiotoxicity.

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