

Destination Healthy Heart:

A ROADMAP TO MANAGING HEART FAILURE



Heart Failure

DUE TO CARDIOTOXINS

NURSE TALKING TIPS SHEET

This nurse tipsheet was developed by AAHFN as resource in facilitating patient education. It provides additional information so that the nurse can supplement their patient teaching with the corresponding patient tipsheet. Because no one page could be exhaustive, a list of resources is provided on page two for additional information.

Patient teaching should focus on:

- Alcohol, cocaine, methamphetamines and other illicit drugs are myocardial depressants and should be avoided. Abstinence often requires referral to a drug rehab program with lifelong commitments to sobriety and to adherence of HF medications.
- Monitor heart function of patients receiving cardiotoxic medications.
- Review all medications the patient is taking including OTCs and supplements
- Encourage exercise as tolerated to strengthen the heart muscle.
- Counsel patient to take all medications as directed; to not stop any medication unless directed by their provider.
- Follow the current AHA/ACC guidelines for management of the patient with heart failure
- Encourage patients to keep all follow up appointments
- Teach patients the symptoms they need to report to their provider.
- Close collaboration among caregivers is also recommended

Background:

- Damage to the heart by toxins is an uncommon but important cause of heart failure.
- Heart failure due to toxins is caused by damage to the heart muscle which leads to impaired pump function.

Causes:

- Common cardiotoxins are listed on the patient sheet.
- Certain chemotherapy agents, chronic alcohol use and use of illicit drugs such as cocaine are toxic to the myocardium.
- The major cardiac complications of chemotherapy are pericarditis, arrhythmias, ischemia and cardiomyopathy.
- There are two main types of chemotherapy-related cardiac dysfunction (CRCD), Type 1 and Type 2.

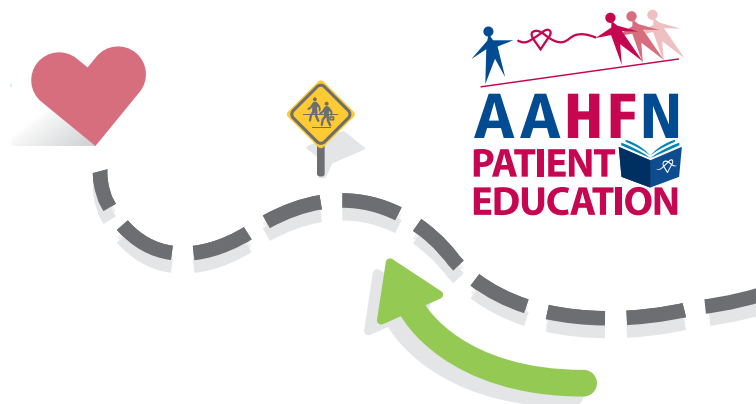
Type I CRCD is caused by anthracyclines which are drugs used to treat a variety of cancers. The most common anthracycline is Adriamycin (doxorubicin). Type I CRCD is thought to be related to an increase in oxidative stress and accumulation of iron in the myocytes. Toxicity is related to the cumulative dose of doxorubicin. Age, female gender and concurrent use of other medications that are cardiotoxic increase the likelihood of CRCD. This type of toxicity is not considered reversible.

Type II CRCD is on the rise due to the use of trastuzumab (Herceptin) to treat HER2 breast cancer. It is thought to be caused by alterations in epidermal growth signaling pathways related to HER2 blockade. The toxic effects of trastuzumab subside with stopping the drug. The risk of Type II toxicity increases when trastuzumab is used with other chemotherapeutics, the patient's age is > 50, or the patient has preexisting heart problems or high BMI. Patients with Type II toxicity are likely to be asymptomatic so a decreased ejection fraction may be seen prior to overt symptoms of heart failure.

- One of the four criteria listed below is needed to diagnose CRCD:
 1. Reduced ejection fraction
 2. Symptoms of heart failure (e.g. shortness of breath, fatigue)
 3. Signs of heart failure (S3, tachycardia)
 4. A decrease in ejection fraction of 5% to < 55% accompanied by signs and symptoms of heart failure, or a decrease in ejection fraction of at least 10% to < 55% without signs and symptoms.

Treatment/Prevention:

- There are no specific guidelines for treatment of chemotherapy induced cardiomyopathy.
- Eliminating the causative toxic agent when possible is essential
- A careful history should be obtained in all patients with signs and symptoms of heart failure.
- Ejection fraction should be assessed at baseline for patients receiving chemotherapy.
- Both ECHO and radionuclide gated ventriculogram may be used for early detection of subclinical diagnosis of cardiomyopathy but there is no consensus regarding the frequency or mode.
- Endocardial biopsy is the gold standard for diagnosing Type I CRCD; data are lacking for Type II CRCD.
- Limiting the total dose of anthracyclines, administering slow IV rather than bolus infusions and administering Dexrazoxane, a chelating agent, may prevent or reduce the toxic effects.
- Due to concern for reducing the antitumor effect, use of Dexrazoxane has been limited to cases where high dose anthracycline therapy is used.
- Treatment for HF due to cardiotoxins is the same as for other heart failure etiologies. Standard care with ACE inhibition, beta blockade, diuresis and aldosterone antagonism is recommended, although cancer patients were not included in trials of these medications.
- Preliminary studies suggest that neurohormonal antagonism may be beneficial; large scale clinical trials are needed.
- Patients with severe heart failure due to chemotherapy may not be candidates for heart transplant as criteria for transplant often requires a person be cancer free for 5 years
- Mechanical circulatory support may be helpful, although data from the INTERMACS Registry suggest these patients may be at increased risk for bleeding.



Bibliography:

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