

# [Congestive cardiac failure with digoxin toxicity](https://assignbuster.com/congestive-cardiac-failure-with-digoxin-toxicity/)

Contents (Jump to)

Criterion-1

Causes, Incidences and Risk Factors of Congestive Cardiac Failure with Digoxin Toxicity:

Comprehensive Understanding of the Disease on Patient and Family:

Criterion-2

Signs & Symptoms

Pathophysiology

Criterion-3

Drug Class

Physiological Effect

Criterion-4

Interventions-Rationales:

Comprehensive Treatment of the Identified Condition:

Supportive care

Electrolyte abnormality management

Bradycardia management

Hemodynamic compromise management

Ongoing monitoring and change of medicine

CASE STUDY ON CONGESTIVE CARDIAC FAILURE WITH DIGOXIN TOXICITY

## Criterion-1

## Causes, Incidences and Risk Factors of Congestive Cardiac Failure with Digoxin Toxicity:

* Digoxin toxicity caused by high levels of digitalis in the body. As in our case study Mrs. Sharon McKenzie, a 77 year old woman, used to take daily 250 mcg of digoxin, which is a very high dose for adult patients. Especially those, who are suffering from congestive cardiac failure, like our patient Mrs. Sharon McKenzie (Neo, et al, 2010). Body receives the therapeutic effect when it stores of 8 to 12 mcg/kg generally with minimum risk of toxicity in most patients with failure of heart and normal sinus or breathing rhythm (Mangoni, 2010).
* People withheart failurewho have this digoxin are commonly prescribed medications called diuretics that remove excess fluid from the part of body. This is also happens that many diuretics can cause potassium loss from the body (Johnson, Inder, Nagle & Wiggers, 2010). Though ultimately it increases the risk of digitalis toxicity. Again, our patient, Mrs. Sharon McKenzie’s potassium level is low; 2. 5 mmol/l. whereas a normal potassium level ranges from 3. 5-5. 0 mmol/l.
* You are more likely to fall into that condition if you take digoxin, digitoxin, or other digitalismedicinesalong with the higher effective drugs that interact withit such as flecainide, quinidine, amiodarone, verapamil, and others. Similarly, Mrs. Sharon McKenzie’s was also taking medication with digoxin like furosemide, warfarin, and enalapril (Siabani, Leeder & Davidson, 2013).
* In recent years the incidence of digoxin toxicity has dropped among patients in hospitals. A study has been done on 183 outpatients, who are receiving on going treatment of digoxin toxicity at 10 urban and rural Department of Veterans Affairs Medical Centers in the Rocky Mountain region, to evaluate whether a similar decline of digoxin toxicity has occurred or not. The statistics over 1-year period, of that study is like that:

Out of the 183 patients:

* 50 (27. 3%) had one or more risk factors for digoxin toxicity.
* Serum digoxin levels were elevated in 13. 6% of patients.
* Hypokalemia in 14. 3%.
* Elevated creatinine levels in 17. 9%.
* And possible drug interactions in 5. 5% of patients.
* The most common risk factor of digoxin toxicity is the patient’s elderly age. Like in our case study, Mrs. Sharon McKenzie is also 77-year old woman. However there are other risk factors too, which render the elderly more vulnerable to digoxin toxicity. These contain an age-related decline in renal function and a decrease in volume of digoxin distribution. There is also an increase in the number of comorbid conditions, including cardiovascular and chronic obstructive pulmonary disease, which heightens vulnerability to digoxin toxicity.

## Comprehensive Understanding of the Disease on Patient and Family:

Digoxin toxicity is a life-threatening condition, and when a serious disease like congestive cardiac failure caused by digoxin toxicity then it can impact severely in a bad way on a patient as well as his/her family (Betihavas, 2011). Due to which his/her family also suffer by seeing their loved one mentally disturbed. Often the patients with CHF who are depressed or who lack social support, the higher the support from the social side the higher the rate of healing as the family and the patient both in complex and double trouble.

## Criterion-2

|  |  |
| --- | --- |
| Signs & Symptoms | Pathophysiology |
| * Severe ventricular arrhythmias:   Sudden cardiac death and loss of consciousness are the basic signs and symptoms of the cardiac arrhythmias. Complaints such as dizziness, lightheadedness, fluttering, dizziness, and pounding, chest discomfort, quivering, shortness of breath, and forceful or painful fast beats are commonly reported with arrhythmias patients. Often, patients notice arrhythmias only after checking their peripheral pulses (Mudge, et al, 2010). | The pathogenesis of the arrhythmias falls into one of two basic mechanisms: increased or covered up automaticity, triggered activity, or re-entry.   * Triggered activity occurs when early after depolarization and delayed after depolarization initiate spontaneous multiple depolarization, precipitating ventricular arrhythmias (Johnson, Inder, Nagle & Wiggers, 2010). * Arrhythmogenesis is probably the most common procedure and results from re-entry. It causes the change of state of mind and mood too. |
| 2) Hyperkalemia:  Higher potassium rate in your blood can affect how your heart works. Symptoms of hyperkalemia can include:   * Abnormal heart rhythm –arrhythmia– that can be life-threatening * Slowheart rate * Weakness (Neo, et al, 2010) | Hyperkalemia may result from an increase in total body potassium secondary to imbalance of intake vs. excretion or from misdistribution between intra- and extracellular space (Nanda, 2009). |
| 3) Hypokalemia:  Usually symptoms of low potassium are mild  Weakness, tiredness, or pain in arms or legs muscles, sometimes this might be so severe to cause inability and disability to move arms or legs due to weakness of muscles (much like a paralysis) (Hughes & Crowe, 2010)   * Tingling or numbness * Nausea or vomiting * Abdominal cramping, bloating * Constipation * Palpitations (feeling your heart beat irregularly) * Urine passing rate is too high simultaneously feeling thirsty mostly (Neo, et al, 2010). | In the heart, low potassium levels make the myositis hypo-polarized or hyper excitable. Thus, arrhythmia occurs as a result of the atrium’s lowered membrane potential due to recovery from inactivation of the Na channel, which may trigger an action potential. In addition to this, reduced potassium in the extracellular space inhibits the IKr potassium current activity, and ventricular depolarization is delayed, which thereby promotes reentrant arrhythmias (Jeon, Kraus, Jowsey & Glasgow, 2010). |
| 4) Neurologic Symptoms:  In the identified condition, the patient may also go through with neurologic symptoms which are: Visual disturbances, disorientation, and confusion. You might experience confusion. Although rare, you might also see bright spots, have blurry vision, or experience blind spots. In addition, you might urinate much more or less than usual (Betihavas, 2011). Your body could also become swollen. | The physiologies of neurological symptoms are not easy to judge and too complex and our getting of them are incomplete mostly. From an evolutionary perspective it is easy to judge the neurological symptoms. Though it makes sense that the genuine physiologies of neurological symptoms are intricate and interrelated (Courtney, et al, 2009). |
| 5) Sinus Node Dysfunction:  Sinus node dysfunction refers to a number of conditions causing physiologically inappropriate atrial rates. Symptoms may be minimal or include weakness, effort intolerance, palpitations, and syncope. Diagnosis is by ECG. Symptomatic patients require a pacemaker.  Sinus node dysfunction includes inappropriate and misbalancing the sinus bradycardia, alternating bradycardia and atrial tachyarrhythmia, sinus pause or arrest, and sinoatrial exit block (Jeon, Kraus, Jowsey & Glasgow, 2010). | SND also causes the abnormalities in SN impulse formation and propagation that also causes abnormalities in the atrium and in the conduction system of the heart (Higgins, et al, 2013). Slow ventricular rates and pauses at the time of stress is the general causes, furthermore, it includes following:   * Fatigue * Angina * Syncope * Dizziness * Fall * Confusion * Heart failure symptoms and palpitations |

## Criterion-3

|  |  |
| --- | --- |
| Drug Class | Physiological Effect |
| 1. Angiotensin-converting enzyme (ACE) inhibitors:   ACE inhibitors cause blood vessels broadness, further descent the amount of work the heart has to do they may also have direct beneficial effects on the heart. These drugs are reducing the symptoms and the need for hospitalization moreover they are helpful to prolong life (Mudge, et al, 2010).   1. Beta-blockers:   Beta-blockers drugs lower down the heart rate and block excessive blockage in the heart. They also helpful in the heart disease. These drugs are usually used with ACE inhibitors and provide an added benefit. They may temporarily worsen symptoms but result in long-term improvement in heart function (Betihavas, 2011). | Although ACE inhibitors improve outcome in patients with systolic dysfunction, many patients with hypertension experience congestive heart failure due to diastolic dysfunction related to left ventricular hypertrophy. ACE inhibitors have been shown to reverse left ventricular hypertrophy in patients with hypertension. A meta-analysis of the effects of several antihypertensive agents suggested that ACE inhibitors were the most effective agent in reducing left ventricular hypertrophy (Katz & Konstam, 2012).  Beta blocker is helpful in improving the function of the failing LV and need to prevent or reverse progressive LV dilation, sphericity, chamber and hypertrophy. Beta blockers also lower down the heart beating rate and LV wall stress. According to recent studies from laboratories have also proven that beta blockers can satisfy cardiomyocyte apoptosis in HF. These are the basic advantages and benefit of beta-blocker for the patient of heart at any higher stage (Katz & Konstam, 2012). |

## Criterion-4

As a registered nurse, my care plan for a patient suffering from Congestive Cardiac Failure with digoxin toxicity would be like, (Driscoll, et al, 2009)

## Interventions-Rationales:

I realize that I would hold the medication – Due to possibility of toxicity

Wait for Electrolytes and digoxin test, as these tests were already ordered for our patient – electrolytes can affect the action of dig and cause dysthymias and to find out the level of dig

Monitor I & O – monitoring for renal function

Monitor for edema and auscultator the lungs

Monitor symptoms, VS – S/E of dig toxicity

Call the doctor. – To get orders to carry out interventions and inform doctor

Start an IV. – For administration of medications (Mudge, et al, 2010).

## Comprehensive Treatment of the Identified Condition:

The main goal of treatment is to correct cardiac toxicity. If the person has stopped breathing, as our patient Mrs. Sharon McKenzie confronting with shortness of breath, startCPRand get emergency medical help (Betihavas, 2011).

Initial treatment includes:

* General supportive care
* Discontinuation of digoxin therapy and prevention of further exposure
* Administration of digoxin-specific antibody fragments (digoxin immune Fab)
* Treatment of specific complications: for example, dysrhythmias and electrolyte abnormalities (Jeon, Kraus, Jowsey & Glasgow, 2010).

## Supportive care

General supportive care includes attaching patients to a cardiac monitor, providing IV fluids in patients with hypotension or volume depletion (with caution for patients with CHF), supplemental oxygen, and/or repletion of electrolytes in patients with electrolyte abnormalities (Mudge, et al, 2010).

## Electrolyte abnormality management

In case of Mrs. Sharon McKenzie, hyperkalemia is only corrected (e. g., with insulin/glucose) if it is considered life-threatening, because of the risk of producing hypokalemia, because her potassium level is low i. e. 2. 5 mmol/l. One study showed that insulin interacts directly with Na(+)/K(+) ATPase pump and alters the effect of digoxin (Betihavas, 2011). This supports the finding that for patients with diabetes, insulin has been shown to have cardio protective effects after digoxin intoxication. Calcium is not used to treat hyperkalemia in patients with suspected digoxin toxicity as it may induce arrhythmia or cardiac arrest.

## Bradycardia management

As Mrs. Sharon McKenzie’s ECG report showed sinus bradycardia, this will be treated with atropine. Atropine can be given every 3 to 5 minutes until there is a response or the 3 mg maximum dose is reached (San Miguel, et al, 2013).

## Hemodynamic compromise management

As Mrs. Sharon McKenzie has signs of hemodynamic insufficiency and/or compromise (e. g., hypotension, altered consciousness or dizziness), digoxin immune Fab is given as primary management (Mudge, et al, 2010).

## Ongoing monitoring and change of medicine

Ideally, digoxin is discontinued and a different medicine for rate control or a different inotrope prescribed (for AF, atrial flutter or CHF, respectively). If the patient has to remain on digoxin for some reason, then the dose of digoxin is adjusted for the patient’s medication profile (Edgley, Krum & Kelly, 2012).

Referencing:

* Jeon, Y. H., Kraus, S. G., Jowsey, T., & Glasgow, N. J. (2010). The experience of living with chronic heart failure: a narrative review of qualitative studies. BMC health services research, 10(1), 77.
* Hughes, J., & Crowe, A. (2010). Inhibition of P-glycoprotein-mediated efflux of digoxin and its metabolites by macrolide antibiotics. Journal of pharmacological sciences, 113(4), 315-324.
* Mangoni, A. A., Woodman, R. J., Gaganis, P., Gilbert, A. L., & Knights, K. M. (2010). Use of nonâ€steroidal antiâ€inflammatory drugs and risk of incident myocardial infarction and heart failure, and allâ€cause mortality in the Australian veteran community. British journal of clinical pharmacology, 69(6), 689-700.
* Siabani, S., Leeder, S. R., & Davidson, P. M. (2013). Barriers and facilitators to self-care in chronic heart failure: a meta-synthesis of qualitative studies. SpringerPlus, 2(1), 320.
* Courtney, M., Edwards, H., Chang, A., Parker, A., Finlayson, K., & Hamilton, K. (2009). Fewer Emergency Readmissions and Better Quality of Life for Older Adults at Risk of Hospital Readmission: A Randomized Controlled Trial to Determine the Effectiveness of a 24â€Week Exercise and Telephone Followâ€Up Program. Journal of the American Geriatrics Society, 57(3), 395-402.
* Nanda, A., Chen, M. H., Braccioforte, M. H., Moran, B. J., & D’Amico, A. V. (2009). Hormonal therapy use for prostate cancer and mortality in men with coronary artery disease–induced congestive heart failure or myocardial infarction. Jama, 302(8), 866-873.
* Edgley, A. J., Krum, H., & Kelly, D. J. (2012). Targeting Fibrosis for the Treatment of Heart Failure: A Role for Transforming Growth Factorâ€β. Cardiovascular therapeutics, 30(1), e30-e40.
* Betihavas, V., Newton, P. J., Du, H. Y., Macdonald, P. S., Frost, S. A., Stewart, S., & Davidson, P. M. (2011). Australia’s health care reform agenda: Implications for the nurses’ role in chronic heart failure management. Australian Critical Care, 24(3), 189-197.
* Mudge, A., Denaro, C., Scott, I., Bennett, C., Hickey, A., & A Jones, M. (2010). The paradox of readmission: effect of a quality improvement program in hospitalized patients with heart failure. Journal of Hospital Medicine, 5(3), 148-153.
* Johnson, N. A., Inder, K. J., Nagle, A. L., &Wiggers, J. H. (2010). Attendance at outpatient cardiac rehabilitation: is it enhanced by specialist nurse referral. Australian Journal of Advanced Nursing, 27(4), 31-37.
* Higgins, R., Navaratnam, H. S., Murphy, B. M., Walker, S., & Worcester, M. U. M. U. (2013). Outcomes of a chronic heart failure training program for health professionals. Journal of Nursing Education and Practice, 3(7), p68.
* Driscoll, A., Davidson, P., Clark, R., Huang, N., &Aho, Z. (2009). Tailoring consumer resources to enhance self-care in chronic heart failure. Australian Critical Care, 22(3), 133-140.
* Neo, J. H., Ager, E. I., Angus, P. W., Zhu, J., Herath, C. B., &Christophi, C. (2010). Changes in the renin angiotensin system during the development of colorectal cancer liver metastases. BMC cancer, 10(1), 134.
* Katz, A. M., & Konstam, M. A. (2012). Heart failure: pathophysiology, molecular biology, and clinical management . Lippincott Williams & Wilkins.
* San Miguel, J. F., Sonneveld, P., Orlowski, R. Z., Moreau, P., Rosiñol, L., Moslehi, J. J., … & Richardson, P. G. (2013). Quantifying the risk of heart failure associated with proteasome inhibition: a retrospective analysis of heart failure reported in phase 2 and phase 3 studies of bortezomib (Btz) in multiple myeloma (MM). Blood , 122 (21), 3187-3187