Congestive cardiac failure with digoxin toxicity

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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
1. Severe ventricular arrhythmias: Sudden cardiac death and loss of consciousness are the basic signs and symptoms of the cardiac arrhythmias. Complaints such as dizziness,	The pathogenesis of the arrhythmias falls into one of two basic mechanisms: increased or covered up automaticity, triggered activity, or re-entry. 2. Triggered activity occurs when early after depolarization

	and delayed
lightheadedness,	after
fluttering,	depolarization
dizziness, and	initiate
pounding, chest	spontaneous
discomfort,	multiple
quivering,	depolarization,
shortness of	precipitating
breath, and	ventricular
forceful or painful	arrhythmias
fast beats are	(Johnson, Inder,
commonly	Nagle & Wiggers,
reported with	2010).
arrhythmias	3. Arrhythmogenesi
patients. Often,	s is probably the
patients notice	most common
arrhythmias only	procedure and
after checking	results from re-
their peripheral	entry. It causes
pulses (Mudge, et	the change of
al, 2010).	state of mind
	and mood too.
2) Hyperkalemia:	Hyperkalemia may
Higher potassium	result from an increase
rate in your blood	in total body

can affect how your heart works. Symptoms of hyperkalemia can include:

4. Abnormal heart rhythm –arrhythmia– that can be life-threatening
5. Slowheart rate
6. Weakness (Neo, et al, 2010)

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,

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

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)

7. Tingling or numbness
8. Nausea or vomiting
9. Abdominal cramping, bloating
10. Constipation
11. Palpitations (feeling your heart beat irregularly)

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).

12. Urine passing rate is too high simultaneously feeling thirsty mostly (Neo, et al, 2010).

4) Neurologic Symptoms:	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
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.

(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.

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:

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).

13. Fatigue
14. Angina
15. Syncope
16. Dizziness
17. Fall
18. Confusion
19. Heart failure symptoms and palpitations

Criterion-3

Drug Class	Physiological Effect
1. Angiotensin-converting	Although ACE inhibitors

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).

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

2. 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).

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).

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