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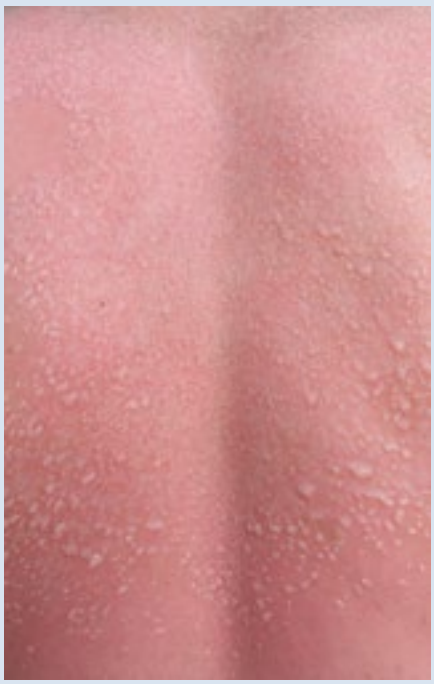

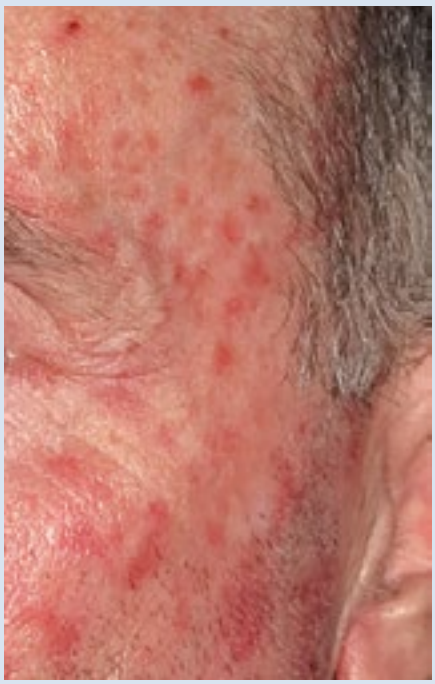

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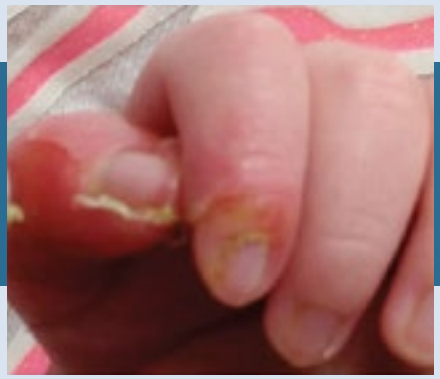

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EXAM SCHEDULE FOR CERTIFICATION & OCC (RECERTIFICATION)

EXAM	EXAM LOCATION	POSTMARK DEADLINE
Family Medicine / OMT Initial Certification Cognitive Exam	Electronic Testing Regional Sites March 19, 2016	October 1, 2015 Late fee through December 1, 2015
Geriatric Medicine CAQ Certification - Cognitive Exam	Electronic Testing Regional Sites March 19, 2016	October 1, 2015 Late fee through December 1, 2015
Geriatric Medicine CAQ OCC/Recertification - Cognitive Exam	Electronic Testing Regional Sites March 19, 2016	October 1, 2015 Late fee through December 1, 2015
Family Medicine / OMT Certification / OCC Performance Evaluation only	San Juan, Puerto Rico ACOFP '16 Convention Convention: April 6 - 9, 2016 Exam: April 4 - 8, 2016	October 1, 2015 Late fee through December 1, 2015
Family Medicine / OMT OCC / Recertification - Cognitive Exam	Electronic Testing Regional Sites May 7, 2016	November 1, 2015 Late fee through December 1, 2015
Pain Medicine CAQ Certification Exam	Chicago, IL May 14, 2016	January 4, 2016 Late fee through February 5, 2016
Family Medicine / OMT Certification / OCC Performance Evaluation only	Anaheim, CA AOA OMED Conference September 16 - 20, 2016 September 16 - 18, 2016	April 1, 2016 Late fee through June 1, 2016
Family Medicine / OMT Initial Certification / OCC Cognitive Exam	Electronic Testing Regional Sites September 24, 2016	April 1, 2016 Late fee through June 1, 2016

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Editor's Message

Dedicated Focus on the Everyday

Amy J. Keenum, DO, PharmD, Editor, Osteopathic Family Physician

This month the articles in OFP range from the common to the rare but with a dedicated focus to a few common conditions we, as family physicians, see every day.

The review article on acute mechanical small bowel obstruction is an easy read. The diagnosis has not changed much over time but it is one of the few conditions for which plain films of the abdomen are still helpful and most patients go on to get a computerized tomographic (CT) scan of the abdomen and pelvis with and without contrast to precisely detect the location of the obstruction and possible underlying causes. It is a surgical condition even if the choice is observation; as such the surgeon should be the one observing.

The side effects of radiation therapy are reviewed and while most of our patients have not had radiation therapy, it is important to keep in mind for those patients who have had radiation treatment. Radiation therapy has changed over the years with the dose being delivered more precisely in both location and dose. Patients who have survived their cancers may present years later with radiation-induced problems. These may include: a secondary cancer, gastrointestinal problems, radiation pneumonitis, pulmonary fibrosis, radiation induced heart disease (RIHD) and radiation dermatitis. Radiation dermatitis is one of the most common side effects of radiation and may be acute and or chronic.

We present an article on a particular rare microbe, lactococcus garvieae, and a broader article on the very common topic of dysuria. Dysuria in non-pregnant females is often cystitis and can be diagnosed and treated symptomatically without urinalysis or culture but beyond that cultures become the tool for diagnosis and treatment. Dysuria may be simple, recurrent or chronic cystitis, pyelonephritis or some less common cause of this symptom not discussed in the article.

As our generally healthy patients flock to the quick clinics, the office seems to be filling with more geriatric patients and that means we will see more congestive heart failure (CHF) in family medicine. It is common in the elderly and sometimes an unlucky younger person will present with this problem. The article reminds us of the most common CHF presentations of shortness of breath on exertion, orthopnea, swelling of the legs and sleep problems. While this is a clinical diagnosis the following may help in the diagnosis and treatment of CHF: chest x-ray, the laboratory test, Brain Naturetic Peptide (BNP) and echocardiograms. The author discusses possible surgical treatments, medical treatments and the important hospital follow up. It is nicely put together and worth your time if you are in clinical practice and have not recently reviewed the topic. We need to reassess patients to see if they are on the most current recommended treatments when they have been our patients for a long time, especially if the severity of illness changes or as they get older.

Happy reading.

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REVIEW ARTICLE TOPICS:

- Abnormal Loss of Weight
- Anemia
- Managing the Problem Opioid Patient
- Nausea with Vomiting
- Osteopathic Consideration in the Infections of the Respiratory Tract
- Osteopathic Principles in Pain Management
- Otitis Media, Acute
- Probiotics: Fact & Fiction
- Sleep Disorders &Treatments

1877-5773X/$ - see front matter.

FROM THE PRESIDENT'S DESK

The "True" Value of Family Medicine

Kevin de Regnier, DO, FACOFP dist.
2015 ACOFP President

A couple of days ago, I spent a ridiculous amount of time arguing with an insurance company trying to get them to pay for the medication I felt my patient needed. A few days earlier, I had to request a conversation with the radiologist because the insurance company algorithm said my patient didn't need an MRI.

I ultimately got both requests approved but my blood was boiling and I was questioning why I became a family physician. Having to continually justify my every action to some insurance clerk is not what I signed up for. Who would want to do this for a living?

I do! One of the great things about being President is that I have the opportunity to talk to students and show them why they too should become osteopathic family physicians. I know that may sound a little ironic given my opening rant, but hear me out.

I believe the future of osteopathic family medicine has never been brighter. For the ninth year in a row, family medicine is the number one recruited specialty.[1] Between 2013 and 2014, average family physician compensation increased 10%, the fourth highest increase of 25 specialties surveyed.[2]

In recent years, policy makers and payers have been asking why the United States spends more money on health care than any other industrialized nation[3] and yet ranks last in the quality of care delivered.[4] According to several researchers, the United States' ranking is dragged down substantially by deficiencies in access to primary care and inequities and inefficiencies in our health care system.[4, 5]

According to Barbara Starfield, MD, MPH from the Commonwealth Fund, the US physician specialty mix is dramatically different than other industrialized nations who have higher quality and lower costs. According to the Commonwealth Fund Primary Care Roundtable 1, Adults (age 25 and older) with a primary care physician rather than a specialist as their personal physician had 33% lower cost of care and were 19% less likely to die.[6]

Primary care physician supply is consistently associated with improved health outcomes (all-cause, cancer, heart disease, stroke, infant mortality, low birth weight, life expectancy, self-rated health). A 12% increase in such physicians (1 per 10,000 population) improves outcomes an average of 4% (range 1.3% to 10.8%; depending on the particular outcome and geographic unit of analysis).[6]

In the United States, an increase of one primary care doctor is associated with 1.44 fewer deaths per 10,000 population. Above a certain level of specialist supply, the more specialists per population, the worse the outcomes. In 35 analyses dealing with differences between types of areas (7) and 5 rates of mortality (total, heart, cancer, stroke, infant), the greater the primary care physician supply, the lower the mortality for 28 analyses whereas the higher the specialist ratio, the higher the mortality in 25 analyses.[6]

It is clear from this data that the solution to America's healthcare crisis is to dramatically increase the number of family physicians and other primary care physicians caring for patients. The remaining question is how to accomplish that goal. We know that family medicine suffers from both an internal and an external identity crisis. Family physicians often rail against the system as I did in my opening paragraphs. Yet of 26 specialties surveyed, family physicians are the most satisfied with their career in medicine.[2]

The key to improving both the image and lot of family physicians is taking this message to those who make healthcare policy in the US. This includes members of Congress, insurance company executives, healthcare leaders, academics, and the public. This is one of the key goals of Family Medicine for America's Health.

FMAH is a joint project of the eight family medicine organizations in the US. Together as FMAH we are taking this message to those who need to hear it. We will no longer be content to wait for the world to "discover" the true value of family medicine. Ultimately, we are in charge of our own destiny, and to me, the future looks very bright.

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2015 ACOFP President Email: president@acofp.org

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REVIEW ARTICLE

Congestive Heart Failure in Adults

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Broward Health Medical Center, Fort Lauderdale, Florida

KEYWORDS:

Heart Failure

Syndrome of Heart Failure

Cardiomyopathy

Heart Failure with Reduced Ejection Fraction

Heart failure is a clinical syndrome that can challenge the primary care physician when other comorbidities are present. Heart failure diagnosis and treatment options are continuously improved by evidenced based medicine. This review article addresses diagnosis and treatment based on the functional classification of heart failure.

INTRODUCTION

Heart failure (HF) is the leading cause of death in the United States. Although the American Heart Association reports that the death rate has decreased for the past few years, there is still a significant number of deaths secondary to heart failure. The first conclusions on risk factors for heart disease were drawn from the Framingham heart study that included 5,209 males from the ages of 30 to 62 from Framingham, Massachusetts. These patients underwent physical exams and lifestyle modifications as a basis for the data. The study later included multiple cohort generations to identify cardiovascular risk factors determined to be important for heart health such as blood pressure, smoking, obesity, diabetes and lack of exercise. More recent studies indicate that heart failure may be due to comorbidities such as chronic obstructive diseases (COPD), obesity, diabetes mellitus (DM), hypertension (HTN) or psychiatric disorders.[1] Studies have shown that there was a risk for increased readmission rates and mortality in patients with coronary artery disease and heart failure; although mortality rates were slightly higher in patients with diastolic heart failure. In patients with systolic heart failure, increased mortality appears to be associated with presence of diabetes mellitus or peripheral vascular disease.[2] Aric study followed 13,150 participants over 17.7 years and it showed that patients with Ankle Brachial Index (ABI) less than 0.90 (hazard ratio: 1.40; 95% confidence interval: 1.12 to 1.74) were at higher risk of developing heart failure.[3]

Framingham risk factors for cardiovascular disease includes age, gender, lipid panel components (cholesterol, low density lipid particle), elevated blood pressure as well as existence of blood pressure treatments. It does not take in account race differences. Although, its applicability should not be extended to heart failure the presence of these factors can help clinicians suspect coronary artery disease as a cause of heart failure.

DEFINITIONS, CAUSES AND PATHOPHYSIOLOGY

The Clinical syndrome of heart failure is characterized by impairment in the ventricular filling, diastolic dysfunction or inability of the heart eject blood or pump failure. Left ventricular dysfunction can occur independent of ejection fraction (EF). Newer classifications by American College of Cardiology Foundation and American Heart Association (ACCF/AHA) are based on ejection fraction and are defined as heart failure with preserved ejection fraction (HFpEF) and heart failure with reduced ejection fraction (HFrEF). However one can take in consideration other echocardiographic parameters when classifying heart failure.

HFrEF is defined by an EF less than 40%. Major causes of heart failure with preserved ejection fraction are coronary artery disease (CAD) with known myocardial infarction (MI), hypertension, diabetes mellitus, metabolic disease, atherosclerotic disease, and peripheral vascular disease (PVD). Borderline HFrEF is defined by an EF between 41% to 49% with attributes, treatments and outcomes similar to HFpEF.

HFpEF is a syndrome characterized by normal ejection fraction and left ventricular diastolic dysfunction observed on echocardiography or cardiac catheterization. The most important risk factor is hypertension.

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The ACCF/AHA stages of heart failure are classified as Stage A, B, C or D. This include patients at risk of heart disease, presence of structural heart disease and absence of symptoms, presence of structural heart disease and presence of symptoms and last refractory heart failure.

The New York Heart Association (NYHA) functional classification encompasses four classes based on exercise capacity and patient'symptoms. This classification represents an independent predictor of mortality and it is tailored to patients with structural heart disease mostly Stages B and C.

Other causes of heart failure are kidney failure, pericardial effusions, myocarditis, arrhythmias and valvular diseases. Endocrine disorders also have been implicated in pathophysiology of heart failure. For example, obesity impacts the workload of the heart secondary to increased circulatory blood volume. Likewise, diabetes mellitus is an independent factor for heart failure. The relationship between Hemoglobin A1c and mortality is "U" shaped with highest mortality at very low or very high Hemoglobin A1c. Patients with thyroid disease can have heart disease secondary to tachycardia and atrial fibrillation related the elevated levels of thyroid hormones. Patients can experience a hypothyroidism related decrease in myocardial contractility and decreased cardiac filling resulting in low cardiac output.

Dilated cardiomyopathy (DCM) refers to dilated ventricles and decreased contractility in absence of hypertension or valvular disease. Dilated cardiomyopathy is not a synonym for nonischemic cardiomyopathy, as the latter can be secondary to hypertension or valvular disease. Familial cardiomyopathies include those who have at least two closely related members with idiopathic cardiomyopathy. *(See Table 1 on page 12).*

HISTORY

A detailed history of present illness usually provides clues to diagnosis of heart failure. Family history may provide information suggestive of a familial cardiomyopathy defined as ≥ 2 relatives with idiopathic DCM. Duration of the symptoms may provide information on ability to recover over time when symptoms are more recent and adaptive processes have not been completed. Symptoms characterization, trigger factors for shortness of breath, presence of chest pain, dyspnea on exertion or exercise intolerance, can all be used in assessment of the NYHA class and clarify if coronary ischemia is the culprit. Symptoms of orthopnea or shortness of breath with recumbent position provide useful information suggestive of elevated left atrial pressures. It occurs secondary to redistribution of blood volume in the pulmonary splanchnic beds. This combination of decreased compliance and inability of left ventricle to pump will result in pulmonary congestion and subsequent symptoms. Orthopnea not improving with chest elevation may be a sign of mitral stenosis. Dyspnea for less than one minute in supine position with absence of symptoms after one minute may be suggestive of pulmonary arterial hypertension. Paroxysmal nocturnal dyspnea is shortness of breath occurring two to fours hours after falling asleep. Left ventricular heart failure is suggested by symptoms relieved with sitting or dangling the feet on the site of the bed. Presence of palpitations may suggest underlying atrial fibrillation or ventricular tachycardia. Decreased exercise tolerance may be difficult to differentiate from physical deconditioning or pulmonary disorders. Gastrointestinal symptoms such as weight loss may indicate cardiac cachexia, which itself carries a poor prognosis. Conversely, fluid overload is implied by weight gain, lower extremity edema, or ascites. Sleep problems may indicate fluid in the lungs, obstructive sleep apnea or pulmonary hypertension. Compliance to medications and diet such as sodium and fluid intake should be investigated.

PHYSICAL EXAM

The physical exam should begin with assessment of Body Mass Index (BMI), blood pressure, and orthostatic changes and continue with palpation of the pulse for strength and regularity. Changes in orthostatic blood pressure may indicate volume depletion or vasodilation. It is important to identify heart sounds such as S3 and signs of fluid overload such as jugular venous distention. S3 represents a low intensity sound heard in early diastole that most of the time is difficult to auscultate. Presence of S4 may indicate diastolic dysfunction or stiff ventricle. Pulmonary crackles, wheezing, decreased breath sounds or Cheynes-Stokes breath sounds consisting of apnea alternating with hyperapnea may occur. Pulmonary rales may not be present in patients with chronic heart failure due to increased lymphatic drainage. Cardiomegaly is suggested by a displaced point of maximal impulse. Hepatojugular reflex may occur and represents a sign of increased jugular venous pressure with palpation of abdomen. Cold lower extremities may indicate decreased cardiac output. Other symptoms include sinus tachycardia, pitting lower extremity edema, hepatomegaly, and possibly an alternating weak and strong pulse suggestive of left ventricular systolic dysfunction. Superimposed functional murmurs of mitral regurgitation or tricuspid regurgitation imply a diagnosis of dilated cardiomyopathy. *(See Table 2 on page 13).*

TABLE 1:
Causes of Cardiomyopathies

Etiology	Detailed Causes	Miscellaneous
Toxic	Cocaine Abuse	Can cause coronary vasospasm and left ventricular dysfunction
	Alcohol	Causes dilated ventricles when there is history of alcohol abuse for more than five years
Medication induced: chemotherapeutic agents	Anthracyclines, taxoids, interferons, and cyclophosphamide	Increase morbidity
Other Toxic Agents	Anabolic steroids, chloroquine, amphetamines, methylphenidate or catecholamines	Used to enchance performance or for weight loss. Ephedra may lead to left ventricular dysfunction and heart failure.
Rate Related	Tachycardia induced cardiomyopathy	Secondary to increased ventricular rate and premature ventricular complexes. Ventricular pacing or right ventricular pacing may result in exacerbation of heart failure.
Connective Tissue Disorder	Systemic lupus erythematosus (SLE), rheumatoid arthritis or scleroderma	Myocardial fibrosis, severe left ventricular dysfunction, conduction abnormalities
Infections	Human immunodeficiency virus (HIV) infections, Chagas disease	DCM Apical aneurysms, mural thrombi, change in configuration of ventricular walls
Noninfectious Causes	Hypersensitivity reactions to medications: Penicillin, Isoniazid, and Phenytoin	Can result in infiltration of myocardium with eosinophils or lymphocytes causing arrhythmias and sudden cardiac death
Peripartum Cardiomyopathies	Unknown Cause	Likely to occur in the third trimester and the risk increases with subsequent pregnancies. The risk of venous thromboembolism is high; and patients will require anticoagulation. Symptoms tend to persist four to six months postpartum, but some patients do not recover their ventricular function.
Other Infiltrative Diseases	Hemochromatosis, amyloidosis or sarcoidosis	May result in heart failure, left ventricular dysfunction, AV blocks, arrhythmia and sudden cardiac death most likely secondary to fibrosis
Stress Induced	Takotsubo cardiomyopathy	Occurs in absence of atherosclerosis. Associated with emotional or physical stress resulting in coronary vasospasm. Patients may experience elevated troponin level and apical ballooning.

TABLE 2:
Differential Diagnosis

Differential Diagnosis	Special Considerations
Pneumonia	Fever, productive cough, Focal signs of consolidation of chest xray, elevated WBC
Asthma	Wheezing on physical exam, can be a symptom of cardiac asthma, usually no other signs of heart failure like S3, JVD, edema
Pulmonary Embolism/DVT	Usually prolonged immobilization, recent surgery or trauma, hemoptysis, pleuritic chest pain, lower extremity edema EKG: sinus tachycardia, S1Q3T3, CT angiogram positive for thrombus
Interstitial Lung Disease	Progressive dyspnea, fine rales on auscultation, high resolution CT:reticular infiltrates, ground glass appearance, honeycombing, restrictive pattern on spirometry
Acute Respiratory Distress Syndrome	Hypoxia, bilateral infiltrates, insert pulmonary artery catheter if cannot differentiate from heart failure with usual diagnostic tests
Pericardial Disease	Chest pain better when leaning forward, Fever, shortness of breath, pericardial friction rub; EKG: electrical alternans, ST elevation with PR depression; pericardial effusion or fibrosis on echocardiogram
Nephrotic Syndrome	Decreased albumin, elevated triglicerides, protein on twenty four hours urine collection >3.5 g
Cirrhosis	Jaundice, hepatomegaly, edema, ascites, abnormal liver function tests, ultrasound liver will show ascites and cirrhotic changes

DIAGNOSIS

Congestive heart failure is a clinical diagnosis. Primary care physician should evaluate carefully history and physical exam and use diagnostic modalities to reinforce clinical diagnosis or to exclude other etiologies. Laboratory data can contribute to diagnosis and help clarify the cause of heart failure. Comprehensive blood count, comprehensive metabolic panel, liver function tests, thyroid function tests, urinalysis, and electrocardiogram should be performed. A decreased hemoglobin and hematocrit can point to high output failure possibility. Elevated blood urea nitrogen and creatinine may be a result of decreased cardiac output or intrinsic kidney injury. An increase in liver function tests may occur secondary to right sided heart failure or hepatic congestion. Thyroid function tests will diagnose hypothyroidism and hyperthyroidism; both diseases can be a primary cause or a contributing factor of heart failure. Urinalysis may show proteinuria suggestive of possible nephrotic syndrome as underlying cause for symptoms. When history, physical exam and laboratory data suggest alternative causes it is reasonable to perform diagnostic tests for hemochromatosis, amyloidosis and pheochromocytoma. If the patient presents with signs and symptoms of heart failure but the diagnosis is unclear, the clinician can verify the levels of Brain Natriuretic Peptide (BNP). An uncertain diagnosis may occur in a patient with superimposed symptoms of COPD. Other noncardiac conditions that can cause high BNP are sepsis, burns, renal failure, age and anemia. Lower levels of BNP have been reported with obesity. BNP levels can be used to guide therapy. However, the TIME-CHF trial failed to show change in quality of life or mortality between BNP guided therapy versus symptom guided treatment despite an augmentation in therapy in BNP group.[4] A BNP level of less than 100 in acute settings and a level of 35 with gradual onset of symptoms levels will exclude heart failure. Levels of troponins may

be elevated in patients with myocardial injury or necrosis, impaired hemodynamics, left ventricular dysfunction and presence of acute coronary syndrome. Congestion should be evaluated with chest radiograph with awareness that patients with chronic heart failure may not show any signs of pulmonary congestion secondary to compensatory effect of the lymphatic system. Echocardiogram will assess left ventricular function, size of atria and ventricles, wall motion abnormalities, wall thickness, valvular disease, inferior vena cava diameter and right ventricular pressure. Patients with high suspicion of coronary artery disease should undergo left heart cardiac catheterization. Hemodynamic assessment with right heart catheterization should be performed when there is unclear volume status, patient is refractory to treatment, has low blood pressures and worsening renal function.

TREATMENT

The primary care physician should be able to manage patients with heart failure. Clinicians should understand evidence based medicine and refer to clinical trials to administer treatments in accordance with population studied and based on common results. Hospitalization should be considered in patients with acute decompensated heart failure, pulmonary edema or cardiogenic shock requiring an acute intervention, intravenous inotropes, mechanical assist devices or hemodynamic monitoring. Referral to cardiology should be in case of suspected underlying coronary artery disease that necessitates interventions, refractory heart failure, or underlying rhythms that may require further testing or procedures. Medical treatment and nonpharmalogical interventions should be focused on close monitoring of symptoms and response or lack of response to therapy. Home environment should be accommodated to such extent that patient has eliminated all foods that might aggravate symptoms of heart failure. A scale should be made available for daily weights. Additional education on diet consisting of salt intake and fluid restriction should be addressed at every visit. Outpatient monitoring of compliance to medications by social services via telephone, mail or text message might be beneficial. Nurse home visits may be adequate to assess environment, perform tests and again monitor adherence to treatment.

Special consideration should be given to elderly patients, as they comprise a large population with heart failure with continual rise in incidence and prevalence of disease. Treatment should be tailored based on comorbidities by avoiding drastic decreases in blood pressure as these changes may increase the risk of falls. Elderly patients already have decreased glomerular filtration rate secondary to aging. Therefore, caution is advised when using diuretics or aldosterone receptor antagonists as they may either worsen renal function or cause cardiac abnormalities due to hyperkalemia. Treatment with digoxin may results in adverse effects in elderly secondary to low body mass index or renal dysfunction. Coadministration of medications such as verapamil, amiodarone, antibiotics (chlarithomycin or erythromycin), or antifungals (itraconazole) may increase digoxin level leading to digoxin toxicity.

STAGE A HEART FAILURE

Stage A heart failure includes patients at risk of heart failure, but without structural heart disease and involves elevated systolic and diastolic blood pressure. For this reason the focus should be placed on controlling hypertension. Medications should be administered to address comorbidities such as diabetes or atherosclerotic disease. In patients with diabetes, blood pressure should be managed with Angiotensin converting enzyme inhibitor (ACEI) or Angiotensin receptor blocker (ARB). A statin should be added to control hyperlipidemia in patients with coronary artery disease or at risk of developing myocardial infarction according to their Framingham score. Risk factors such as obesity, smoking, alcohol, cocaine and amphetamine use should also be addressed. Additionally, patients should be counseled at every visit about the risks associated with their habits.

STAGE B HEART FAILURE

Stage B heart failure includes patients with structural heart disease, but without signs and symptoms of heart failure. Patients with a family history of cardiomyopathy, long standing history of hypertension, prior myocardial infarction or receiving cardiotoxic agents should be evaluated with an echocardiogram. Studies such as COPERNICUS and CAPRICORN compared carvedilol to placebo and showed the benefit of beta blockers (BB) in heart failure. Capricorn trial showed a 23% relative reduction in mortality when medication is started 3-21 days after myocardial infarction (MI) in patients with reduced left ventricular function <40%.[5] The Copernicus trial showed a 35% relative risk reduction in mortality. In both trial patients were also receiving ACEI.[6]

Patients with low EF, but without symptoms of heart failure should receive treatment with ACEI or ARBs and BB. SAVE trial randomized 2231 patients to Captopril and placebo and showed a reduction in mortality in patients with MI and LVEF less than 40% without evidence of overt heart failure.[7] Val-HeFT trial randomized valsartan to placebo and showed no mortality benefit when added to ACEI or BB. There was statistical significant reduction in a morbidity associated with heart failure.[8] VALIANT study showed that Valsartan was non-inferior to Captopril in patients with heart failure, but failed to show a mortality benefit. If a patient has DM and

HTN consideration should be given to ACEI or ARBs. In a patient with refractory hypertension, spironolactone can be added to the regimen.[9] RALES trial included 1663 patients NYHA Class III-IV with EF less than 35% and randomized patients to 25 mg spironolactone daily and placebo. Trial demonstrated a 30% reduction in mortality, progressive reduction in heart failure, rate of hospitalization and sudden cardiac death.[10] EMPHASIS- HF trial randomized Eplerenone to placebo and included 2737 patients with NYHA class III-IV and left ventricular EF less than 30%. The study showed a 34% reduction in the risk of death from CV causes and hospitalization for HF.[11]

Newer trials such as PARADIGM-HF examined the effect of Neprilysin in patients with heart failure. Neprilysin is an endopeptidase that breaks down peptides such as BNP, bradykinin, and adrenomedullin contributing to decreased remodeling, vasoconstriction and sodium retention. "LCZ696" is the resulting product of combining angiotensin receptor neprilysin inhibitor (ARNI) such as neprilysin sacubitril with valsartan. Patients included in the trial displayed NYHA class II-IV symptoms, left ventricular ejection fraction less than 40% and were receiving beta blockers and ACEI. Patients were randomized to a single blind run-in period, during which all patients received enalapril for 2 weeks. Thereafter, medication was stopped for 1 day then all patients received ARNI for 4-6 weeks. Following run-in phase patients were randomized to ARNI and enalapril. Upon a 27 month follow up a reduction in mortality was noted in ARNI group as well as reduction in cardiovascular mortality and HF hospitalization. However, the risk of adverse effects were higher in ARNI group.[12]

STAGE C HEART FAILURE

Majority of the patients that present to emergency room are Stage C heart failure meaning they are symptomatic and have structural heart disease. This category of patients should monitor their weight, salt intake, exercise (if their functional status allows) and take medications as prescribed. A regimen consisting of ACEI or ARBs and BB. should be administered. Consideration should be given to volume status; while overload should be treated with diuretics. In patients of African American race, hydralazine and nitrates in combination are advised.

Aldosterone antagonists presents a choice if patients have NYHA Class II-IV symptoms and GFR greater than 30; or potassium levels less than 5.0. Diuretics should be added to standard regimen if there is evidence of fluid retention. Caution is advised when ACEI are prescribed and patients experience SBP less than 80, creatinine is greater than 3.0, potassium greater than 5.0 or there is bilateral renal stenosis. Contraindications to ACEI include angioedema and pregnancy. ARBs have demonstrated reduced hospitalization and mortality in heart failure and remain an alternative in patients intolerant to ACEI experiencing cough. Patients experiencing angioedema while on ACEI should not be started on ARBs as some patients can develop angioedema while on latter medication.

Beta blockers such as Carvedilol, Bisoprolol and Metoprolol have shown reduced mortality and hospitalization. The Seniors trial included patients with HFpEF that showed that Nebivolol can cause a small reduction in mortality in elderly patients.[14] Beta blockers can be used in patients with reactive airway disease or low heart rate if they are asymptomatic. Doses should be adjusted if patient becomes bradycardic with associated dizziness or second or third degree Atrioventricular (AV) block. Beta blockers should be discontinued when patient becomes hypotensive. Aldosterone receptors antagonists have been shown to reduce mortality in patients with HFrEF according to RALES trial, EPHESUS and EMPHASIS-HF trials.[10, 11, 13] Creatinine should be at a level of less than 2.0 in females and less than 2.5 in males, while potassium should be less than 5.0 at initiation of treatment. Potassium levels can be monitored within two to three days then at seven days.

Most available data shows that Hydralazine and isosorbide dinitrate have been effective in reducing mortality, but not the rate of hospitalizations. Combination regimen represents an alternative in patients unable to take ACEI or ARBs secondary to renal insufficiency, hypotension or allergic reaction. Hydralazine/isosorbide dinitrate should be used in the African American population and in conjunction with ACEI or ARB and aldosterone antagonists as it has shown in clinical trials to increase survival in patients with NYHA Class III-IV.[15] Treatment should start at 37.5 mg hydralazine and 20 mg tid isosorbide dinitrate titrate up to a maximum of 225 mg hydralazine and 120 mg of isosorbide dinitrate. V-HePT trial in 1984 randomized hydralazine/isosorbide to prazosin and placebo, but failed to show a survival benefit when compared the three groups.[8] A-HEFT trial showed a 40% reduction in mortality when combination drug was added to standard therapy consisting of ACEI, BB and diuretics.[15]

Digoxin reduces rate of hospitalization, but has no impact on mortality.[17] It should be considered in patients with heart failure with persistent symptoms despite use of ACEI and aldosterone antagonists. Practitioners should not institute treatment with digoxin in patients with AV block unless a pacemaker is present. Be cautious when a patient is on medications that can depress AV node, such as amiodarone or BB. However, studies have shown that digoxin works well in conjunction with BB to control ventricular response in patients with atrial fibrillation and heart failure. Digoxin toxicity occurs at levels greater than 2 ng/ml or at lower levels

of digoxin with low potassium or low magnesium as well as in patients with hypothyroidism, low lean body mass or renal dysfunction.

Administer anticoagulation in patients with HFrEF with atrial fibrillation having the following risk factors: hypertension, diabetes mellitus, prior stroke or TIA, and an age greater than 75 as calculated by CHADS2 score. Always consider that risk of thromboembolic stroke is 1-3% even in patients with low EF and intracardiac thrombi. The pathophysiology of increasing risk of cerebrovascular accident is related to the stasis of the blood in hypokinetic chambers and blood vessels leading to increased activity of procoagulant factors. Anticoagulation with warfarin or newer anticoagulants such as Rivaroxaban, Apixaban and Dabigatran should be initiated when patients with heart failure and atrial fibrillation, and at least one risk factor identified by CHADS2 score. The newer anticoagulants do not require INR monitoring which may be overwhelming to patients. The data is insufficient regarding use of aspirin in patients with heart failure, but no evidence of atherosclerotic disease manifested as prior myocardial infarction or coronary artery disease.

Statins should not be used in patients with heart failure without evidence of atherosclerotic disease. The Corona trial observed the role of Rosuvastatin in patients with ischemic heart failure, NYHA class II-IV, and failed to show a difference between placebo and rosuvastatin in primary end points such as cardiovascular mortality, non-fatal MI and nonfatal stroke. There was a reduction in hospitalization secondary to cardiovascular causes in patients on rosuvastatin.[18] The Gissi-HF study observed patients with ischemic and nonischemic HF while taking Rosuvastatin and failed to show a change in clinical outcomes.[19]

Omega-3 polyunsaturated fatty acids (PUFA) have not reach statistical significance in reduction of all cause mortality in HISSI-HF trial.[20] A total of 6975 patients with NYHA class II-IV patients were randomized to 1 gm of PUFA and placebo. There was a borderline statistically significant reduction in time to death or admission to hospital for heart failure. Nutritional supplements are not recommended for treatment of heart failure except to replenish deficiencies.

Antiarrhythmics such as Class I and class III sotalol and dronedarone should be avoided in patients with heart failure. Amiodarone and dofetilide can be used because of a neutral effect on mortality. Calcium channel blockers have negative inotropic effect and can slow conduction in calcium dependent sinoatrial (SA) node and AV node. Both should be avoided in heart failure. The Praise trial demonstrated that amlodipine causes no harm in patients with HF NYHA class III-IV with ejection fraction less than 30%.[21]

NSAIDS inhibit synthesis of prostaglandins resulting in vasoconstriction, reabsorption of salt in loop of Henle and collecting tubule and water retention exacerbating the symptoms of heart failure.

AUTOMATIC IMPLANTABLE CARDIOVERTER DEFIBRILLATOR (AICD)

The primary care physician should be aware of risk of sudden cardiac death in patients with heart failure and appropriately refer patients to electrophysiologist for further therapy specially when practicing in a rural area with decreased referral ability. The population who benefits from AICD placement includes patients at risk of recurrence of symptoms with associated ventricular tachycardia, ventricular fibrillation, history of sudden cardiac death and unexplained syncope. There is no net benefit in using ICD in patients within 40 days after acute MI. Madit-I and Madit-II showed a mortality benefit in patients with LVEF less than 35% at 40 days post-MI and NYHA Class II and III or patients with LVEF less than 30% at 40 days post-MI and NYHA Class I.[22, 23] Definite trial compared ICD and standard therapy and demonstrated a reduction in mortality in patients with nonischemic cardiomyopathy and LVEF less than 35 %, but it did not reach statistical significance. This trial showed a reduction in sudden cardiac death from arrhythmias that reached statistical significance.[24]

Patients with HFrEF should undergo goal directed medical therapy for three to six months followed by an echocardiogram to assess LV function. Although ICD seems to be a great device in reducing the risk of sudden cardiac death from ventricular arrhythmias, patients might have a decreased quality of life secondary to frequent shocks that may lead to a post-traumatic stress syndrome. It would be reasonable to administer antiarrhythmics and perform a catheter ablation in that situation, which may decrease the need for ICD shocks.

CARDIAC RESYNCHRONIZATION THERAPY (CRT) FOR VENTRICULAR PACING

The benefit of biventricular pacing is seen in improvement of pathophysiology in patients with heart failure and manifests as decreased remodeling of the heart, improved contractility of the ventricle, diminished secondary mitral regurgitation and improved EF. Indications for CRT pacing include mostly patients with HF NYHA class III and IV, wide QRS and left bundle branch block (LBBB). BLOCK HF trial randomized patients to right ventricular pacing and biventricular pacing. In patients with reduced EF biventricular pacing increased left ventricular end systolic volume index and decreased death and urgent care visit for heart failure.[25]

In patients with characteristics such as NYHA class I-II heart failure symptoms, EF less than 30 % and QRS greater than 130 ms, trials have showed better outcomes when treated with CRT-D alone when compared to ICD alone.[26] RAFT trial included patients with HF NYHA Class II-III and showed reduction in mortality with CRT. The role of CRT in HF NYHA Class I is unclear based on studies that have showed a reduction in hospitalizations with no difference in mortality.[27]

STAGE D HEART FAILURE (REFRACTORY)

Signs and symptoms of Stage D heart failure include cardiac cachexia, worsening renal function, low blood pressures, intolerance to ACEI or BB due to hypotension and persistent dyspnea. Other signs include increased use of diuretics to greater than 160 mg per day, addition of metolazone therapy and increased ED visits to greater than two per year for heart failure. Other causes for weight loss or dyspnea should be investigated. Workup should be sought for thyroid and pulmonary disorders as causes of worsening of the symptoms. Patients should be evaluated for medication compliance as another cause of treatment failure. Patients with stage D heart failure should undergo fluid restriction of 1.5-2 L daily. Fluid restriction with salt restriction may provide a better efficacy of diuretics. Limiting salt will result in better excretion of the water and less activation of vasopressin, which physiologically mediates water reabsorption in distal tubule. Patients experiencing cardiogenic shock should receive inotropic support, although inotropes have not demonstrated improved outcomes. Milrinone causes inhibition of phosphodiesterase 3 resulting in improved diastolic relaxation and vasodilation of arteries. Dobutamine and dopamine is involved in stimulation of adrenergic and dopaminergic receptors respectively. Patients with hypotension and decreased systemic perfusion, low cardiac index and systolic dysfunction will need inotropic support. In patients refractory to inotropic support, mechanical circulatory support can be used as a bridge to transplant or to candidacy for transplant if patients meets criteria for heart transplantation. Mechanical support devices are available in a variety of forms such as percutaneous or surgical. Such devices can assist the right, left or both ventricles, can provide continuous or pulsatile flow and can be used for short and long term management. These devices have shown an improvement in survival and functional capacity. Mechanical support devices are used as therapy in patients with pulmonary hypertension and heart failure who are not candidates for transplant due to irreversible elevated pulmonary arterial pressures. With the help of such devices, patients may become eligible for transplant over time. The REMATCH trial randomized 129 patients in end stage heart failure who were not eligible for transplant due to optimal therapy. The trial showed an improved mortality in patients with left ventricular assist device with an absolute risk reduction of 28.5%. The median survival of patients with Heartmate device was 408 days compared to 150 days in patients receiving standard therapy for heart failure. Patients included in the trial received inotropic support.[28]

Cardiac transplantation is considered the main treatment of the Stage D heart failure. Indications for heart transplantation includes patients with hypertrophic cardiomyopathy, reversible pulmonary HTN, peripartum cardiomyopathy and restrictive cardiomyopathy. Statistical analysis have shown better outcomes in these groups of patients.

ACUTE DECOMPENSATED HEART FAILURE

Hospitalized patients with heart failure can experience an acute coronary syndrome, accelerated hypertension, an acute decompensated heart failure, cardiogenic shock or acute right heart failure. A physician should assess the hemodynamics by looking at blood pressure, central venous pressure, degree of congestion (both wet and dry), and degree of perfusion hot or cold. Chest radiography should help diagnosis, but a negative chest X-ray does not exclude heart failure. BNP levels should guide treatment when diagnosis is uncertain. Levels should be adjusted for age, while low levels should not defer diagnosis when clinical picture is strongly suggestive of heart failure. Practitioners will identify causes that promoted the acute decompensation such as an acute coronary syndrome guided by electrocardiographic changes or troponin levels. Factors that may contribute to acute decompensation of heart failure include the following: medication noncompliance, ischemia, elevated blood pressure (especially in African American males and patients with HFpEF), atrial fibrillation, medications with negative inotropic support or medications that increase salt retention such as steroids, pulmonary embolus, alcohol, cocaine or methamphetamines, DM, and infections or valvular disease endocarditis.

On admission, oral medications will be continued and up titrated in absence of hemodynamic instability. ACEI and beta blockers should be reduced or withheld if patients manifest worsening renal failure or hypotension, marked volume overload or low cardiac output. Diuretics in patients with heart failure have shown to decrease morbidity. Patients with heart failure should be treated with diuretics to relieve congestion and in fashion to not cause a rapid decrease in intravascular volume. Electrolytes and daily weights, strict fluid intake and outtake should be monitored closely. DOSE trial evaluated the effects of administration of continuous infusion versus intermittent boluses with low dose and high dose diuretics in patients with heart failure. The high dose diuretic consisted of 130 mg IV bid. Notably, the Dose trial revealed that patients included in the study were already receiving a dose of 80 to

240 mg daily of furosemide for at least one month prior to hospitalization. The study did not show any improvement between study groups regarding global relief of symptoms or change in creatinine level. As secondary end point, high dose diuretics were associated with greater relief of dyspnea, change in weight, or net fluid loss at 12 hours. These differences were not seen with either intermittent boluses or continuous infusion of diuretics.[29] Currently, there are no studies showing the effect on mortality. If patients remain refractory to diuretics use after addition of a second diuretic, they should undergo assessment of filling pressures and cardiac output with right heart catheterization. A treatment option would be adding a low dose dopamine infusion to loop diuretics after all options have been exhausted. If these treatments are unsuccessful, patients should undergo treatment with ultrafiltration. UNLOAD and CARRESS-HF trials used different approaches to ultrafiltration. Both studies included critically ill patients refractory to treatment. CARESS-HF showed a change in creatinine level at 96 hours, but failed to show a change in clinical well being of the patient or reduction in dyspnea.[30] UNLOAD trial randomized 200 patients to ultrafiltration and IV diuretics showed an improvement in weight at 48 hours but failed to show an improvement in dyspnea score or length of hospitalization.[31] Patients with acute decompensated heart failure should receive nitroglycerin, nitroprusside or nesiritide in absence of hypotension. Nitroglycerin will decrease the venous preload and reduce pulmonary congestion. Patients with HF and hypertension, coronary ischemia and mitral regurgitation should be considered for administration of nitroglycerin. Sodium nitroprusside effects preload, afterload and relaxes pulmonary vasculature, but has the potential to cause hypotension. Thus, it is an excellent choice for patients with vascular congestion and significant mitral regurgitation affecting the left ventricular function.

ANTICOAGULATION

Patients with heart failure should receive prophylaxis for deep vein thrombosis, as decreased cardiac output, and venous stasis can promote clot forming. Treatment consists or Enoxaparin (Lovenox) 40 mg subcutaneous daily or Heparin 5000 units subcutaneously every eight hours. Graded compression stocking should be used with caution in patients with heart failure as they may contribute to cutaneous complications.

CARE AFTER HOSPITALIZATION

Upon discharge, patients should be enrolled in multidisciplinary heart failure disease management programs, and a follow up appointment should be scheduled within 7-14 days to reduce the risk of hospital readmission. Additionally, a telephone follow up within three days from discharge should address adherence to medications and change in behaviors. Cardiac rehabilitation has been shown to improve mortality, rate of hospitalization and functional capacity. Patient should be referred for supervised exercise training following discharge.

COMORBIDITIES

Comorbidities such as Atrial Fibrillation and Heart Failure as well as surgical considerations in heart failure

ATRIAL FIBRILLATION AND HF

Atrial fibrillation predisposes heart failure. However, heart failure is an independent risk factor for atrial fibrillation. Assessment should begin with history and physical examination to assess duration and type of atrial fibrillation. CHADS2-Vasc score represents an assessment tool used by physicians to determine need for anticoagulation in patients with atrial fibrillation. Components of CHADS-Vasc score include: congestive heart failure, HTN, age greater than 65, DM, stroke, vascular disease, age greater than 75 and sex. A score of 1 is assigned to all components except stroke or age greater than 75, which receives a score of 2. Score 0 does not require anticoagulation. Score 1 aspirin or other forms of anticoagulation can be considered based on a mutual decision between physician and patient. Score of 2 or greater requires anticoagulation as risk of stroke increases considerably. Patients should have an EKG to assess rhythm, left ventricular hypertrophy, bundle blocks, prior MI and atrial arrhythmias. Furthermore, an echocardiogram is warranted to assess for valvulopathy, right and left atrial size, right ventricular systolic pressure, atrial thrombus, left ventricular hypertrophy and pericardial disease. Laboratory data is necessary to assess thyroid and liver function tests, as well as electrolytes, BUN and creatinine. A transesophageal echocardiogram, electrophysiologic study or chest x-ray may be needed based on clinical presentation and results of above mentioned tests.

Patients who develop heart failure secondary to atrial fibrillation should undergo a rhythm control approach to improve their atrial preload. A newly diagnosed heart failure in presence of atrial fibrillation with rapid ventricular response can be addressed with a rate control therapy and assess response to therapy by monitoring ejection fraction. A second approach can be focused on rhythm control therapy consisting of amiodarone and cardioversion after one month. Patients with heart failure who develop atrial fibrillation can receive treatment with a rate control agent or an antiarrhythmic since both approaches seem to be non-inferior. Improvement with antiarrhythmics is observed when patients underwent catheter ablation. Practitioners should remember that beta blockers have a mortality reduction benefit. Digoxin can be added to treatment with beta blockers or calcium channel blockers. Procedures such as catheter ablation and CRT placement should be considered when atrial fibrillation is refractory to treatment with antiarrhythmics.

Patients with heart failure with preserved ejection fraction and atrial fibrillation typically experience shortened diastolic filling time and loss of atrial kick to left ventricular diastolic filling. These patients can be treated with ACEI or ARBs and beta blockers.

SURGERY AND HEART FAILURE

Patients with heart failure should undergo revascularization in the presence of 50% stenosis of the left main or 70% stenosis of coronary arteries and EF 35-50%. Surgical intervention may include mitral or aortic valve replacement, septal myectomy for hypertrophic cardiomyopathy or ablation for ventricular arrhythmias. STICH-1 trial included 1212 patients with LVEF less than 35% randomized to CABG versus optimal medical therapy showed no mortality benefit from any cause but there was a decrease in mortality and hospitalization from cardiovascular events. This trial excluded patients with left main stenosis greater than 50%.[32] STICH-2 trial failed to show a mortality benefit in patients who underwent surgical ventricular reconstruction in addition CABG to CABG alone.[33]

Heart failure with preserved ejection fraction has not been addressed much by current clinical trials. Little is know about effectiveness of standard medications in this population. The TOPCAT trial randomized 3,445 patients to spironolactone and placebo. Patients included had LVEF greater than 45%, findings of heart failure or elevated levels of BNP. There was no difference in primary outcome regarding CV mortality. A decrease in hospitalizations in patients in North America was noted, but the finding was not consistent in sample population from Eastern Europe. Geographic disparities might be secondary to overdiagnosis of heart failure or variability in heart failure practices.[34]

Although heart failure diagnosis and treatment approach may be a challenging task to the practitioner, the goal of treatment of heart failure is to control BP, reduce symptoms and risk factors in order to prevent morbidity. This goal can be achieved by providing a medication regimen individualized to a particular patient.

TABLE 3:

Summary of Treatment

Stages of Heart Failure	Treatment	Goals
Stage A	ACEI, ARBs, Statins	Treat HTN, DM, obesity
Stage B	ACEI, ARBs, BB, Statins ICD if apppropriate	Prevent further cardiac remodeling
Stage C	Diuretics, BB, ACEI or ARBS, Aldostorone antagonists, Hydralazine/Isosorbide dinitrite if African American race, Digoxin, ICD or CRT if appropriate	Address HTN, DM, obesity Symptoms control, prevention of hospitalization; decrease mortality
Stage D	Heart transplant, Ventricular assist device, Palliative care, Hospice, Inotropes, ICD deactivation	Symptoms control, Decrease rate of hospitalizations, End of life care
Acute Decompensated Heart Failure	Diuresis, If unsuccessful add second agent, or consider ultrafiltration; Inotropes: Dopamine, Dobutamine, Milrinone; Nitroglycerin if HF and HT, coronary ischemia or mitral regurgitation; Continue ACEI and BB if tolerated or reintroduce as soon as patients tolerates, DVT prophylaxis; If hyponatremia consider Tolvaptan; Noninvasive ventilation: CPAP or BIPAP	Find cause or precipitant; Dopamine low dose as adjunct to diuresis to improve renal function; Pulmonary artery catheter does not improve survival; Milrinone: causes peripheral and pulmonary vasodilation, do not administer unless patient is in shock; Dobutamine use in cardiogenic shock, increases heart rate and oxygen demand

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REVIEW ARTICLE

The Clinical Management of Acute Mechanical Small Bowel Obstruction

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KEYWORDS:

Small Bowel Obstruction

Conservative Management

Acute mechanical small bowel obstruction (AMSBO) is a common emergency and a significant cause of hospitalization. Due to the variation in small bowel obstruction-related symptomatology, many patients are unaware of the seriousness of their clinical condition and do not seek immediate medical attention. Consequently, such patients forego a visit to the hospital emergency department and often present to their primary care physician (PCP). PCPs, with hospital admitting privileges, and other hospital-based physicians, must have a sound understanding of the principles underlying the treatment of AMSBO. All patients with suspected AMSBO should be hospitalized and treated initially with conservative management. This includes bowel rest with early decompression, fluid resuscitation, and correction of electrolyte abnormalities. Water-soluble contrast medium can be useful adjunct in this approach; it has both diagnostic and therapeutic purposes. Furthermore, water-soluble contrast medium is safe and reduces the need for surgery, time to resolution and hospital stay. Non-operative management can be prolonged up to 72 hours in the absence of strangulation or peritonitis. In contrast, ambulatory patients presenting with ominous clinical signs and symptoms should be considered for immediate surgical intervention. Indications for surgery include strangulation, peritonitis, intractable vomiting, complete or closed loop bowel obstruction, or failure to improve after 72 hours of conservative management.

INTRODUCTION

Small bowel obstruction occurs when the movement of intraluminal contents within the intestine is restricted, leading to a partial or complete obstruction. The factors that lead to obstruction originate from either a mechanical or a functional pathophysiology. Mechanical obstructions can occur outside of the intestine (extrinsic), within the intestinal wall (intrinsic), or within the lumen. The most common causes of mechanical small bowel obstruction are postoperative adhesions and hernias. Postoperative intra-abdominal adhesions are the etiology in up to 75% of cases of small bowel obstruction.[1] Although tumors, strictures, gallstones and foreign bodies are also possible causes, they are less common. Functional small bowel obstruction, or adynamic ileus, can manifest with symptoms similar to mechanical obstruction and may be difficult to differentiate without appropriate clinical evaluation. Since adhesions are the most common cause of small bowel obstruction, we will review the proper diagnostic evaluation & initial management of AMSBO, and subsequently discuss the indications for surgical referral.

DIAGNOSTIC EVALUATION

Early diagnosis of small bowel obstruction is essential for successful medical management. This is especially important for AMSBO which may lead to intestinal strangulation, a surgical emergency that can result in bowel ischemia, necrosis and perforation. Ischemia complicates 7 to 42 percent of bowel obstruction and significantly increases mortality.[2] Diagnostic evaluation should focus on differentiating mechanical from functional obstruction *(Table 1)*, determining if the obstruction is partial or complete, and discriminating simple from strangulating obstruction. An appropriate assessment will guide the clinician in determining whether AMSBO should be treated with conservative medical management or surgery.

TABLE 1:

Differential Diagnosis of Bowel Obstruction

Mechanical	Functional
Adhesion	Ileus
Hernia	Pseudo-obstruction
Tumor	Intra-abdominal Sepsis
Bowel Inflammation	Pneumonia
Gallstone	Electrolyte Imbalance
Volvulus	
Intussusception	

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Initial assessment should begin with elicitation of pertinent history, such as prior abdominal operations, which may suggest the presence of adhesions. The most important risk factors for adhesions are type of previous surgery and the extent of peritoneal damage obtained during surgery. For example, surgeries of the rectum and colon are associated with a higher risk of adhesion-related problems.[3] Total colectomy with ileal anal pouch anastomosis has the highest incidence of adhesion-related problems, followed by gynecological surgeries and open colectomies.[3] Other risk factors for small bowel obstruction include age younger than 60, previous laparotomy within 5 years, and history of peritonitis, multiple laparotomies, emergency surgery, omental resection, or penetrating abdominal trauma.[4] In addition, identification of prescription medications, such as narcotics, and recreational drugs, that have the potential for disrupting bowel function, could prove insightful.

Bowel obstruction usually presents with abdominal pain, nausea and vomiting, absence of flatulence or stool, abdominal distention, and dehydration.[5] Abdominal distention is more pronounced if the obstruction localizes to the proximal jejunum or beyond. Proximal obstructions tend to present with more frequent cramps compared to distal obstructions, which present with cramps of longer duration and usually with less severity.[6] Important clinical signs and symptoms, including tachycardia, fever, localized pain or peritonitis, and leukocytosis, may indicate strangulation of the bowel. Physical examination must include a thorough inspection for any external or internal hernias. External hernias that often obstruct bowel include hernias in the inguinal canal, femoral canal, and previous sites of incision. Internal hernias are related to congenital mesenteric defects, obturator foramen hernia, and improper closing of mesentery created by previous surgeries.[7] The presence of gross or occult blood rectally may be suggestive of intestinal strangulation.

Supine and erect abdominal plain films should be obtained in the diagnostic work up of suspected small bowel obstruction. A complete abdominal series can be as sensitive as a Computed Tomography (CT) scan in detecting high grade bowel obstruction (86% vs 82%).[8] However, plain films are less useful for detecting low grade or partial bowel obstruction, with a sensitivity ranging from 70 to 86%.[9] Despite limitations in sensitivity, plain films should be considered as they are widely available and less costly.

When plain films are inconclusive, CT scan can be highly diagnostic, with a sensitivity of 80% to 90% and specificity of 70% to 90%.[9] Findings of small bowel obstruction on a CT scan often include a proximally dilated bowel with a discrete transition zone leading to decompressed bowel distally; contrast will often not pass through the transition zone. A CT scan can confirm the presence of complete obstruction and identify the cause. Furthermore, it can confirm the presence of strangulation with a sensitivity & specificity higher than 90% and a negative predictive value of nearly 100%.[10] A CT scan should be considered when clinical history, physical examination, and plain films are inconclusive in making the diagnosis of AMSBO obstruction.[4]

Small bowel obstructions that are partial or low grade are less likely to be detected with CT scan. In particular, subtle transition zones as well as unsuspected closed loop obstructions can be difficult to diagnose. In such cases, small bowel follow through studies coupled with CT has a much higher diagnostic yield, with sensitivity and specificity each approaching 100%.[11] These studies are often more labor intensive but provide greater sensitivity in detecting luminal and mural etiologies of obstruction.

Water-soluble contrast medium, such as Gastrografin, can also be useful in evaluating AMSBO. In particular, radiographs utilizing water-soluble contrast medium can be predictive of the need for surgery in patients with adhesive small bowel obstruction.[12] Partial bowel obstruction is likely to resolve with conservative treatment if water-soluble contrast medium is detected radiographically in the colon. In contrast, absence of contrast medium in the colon after 24 hours is indicative of complete bowel obstruction and surgical treatment will likely be required. Water-soluble contrast medium administration has both diagnostic and therapeutic value.[12]

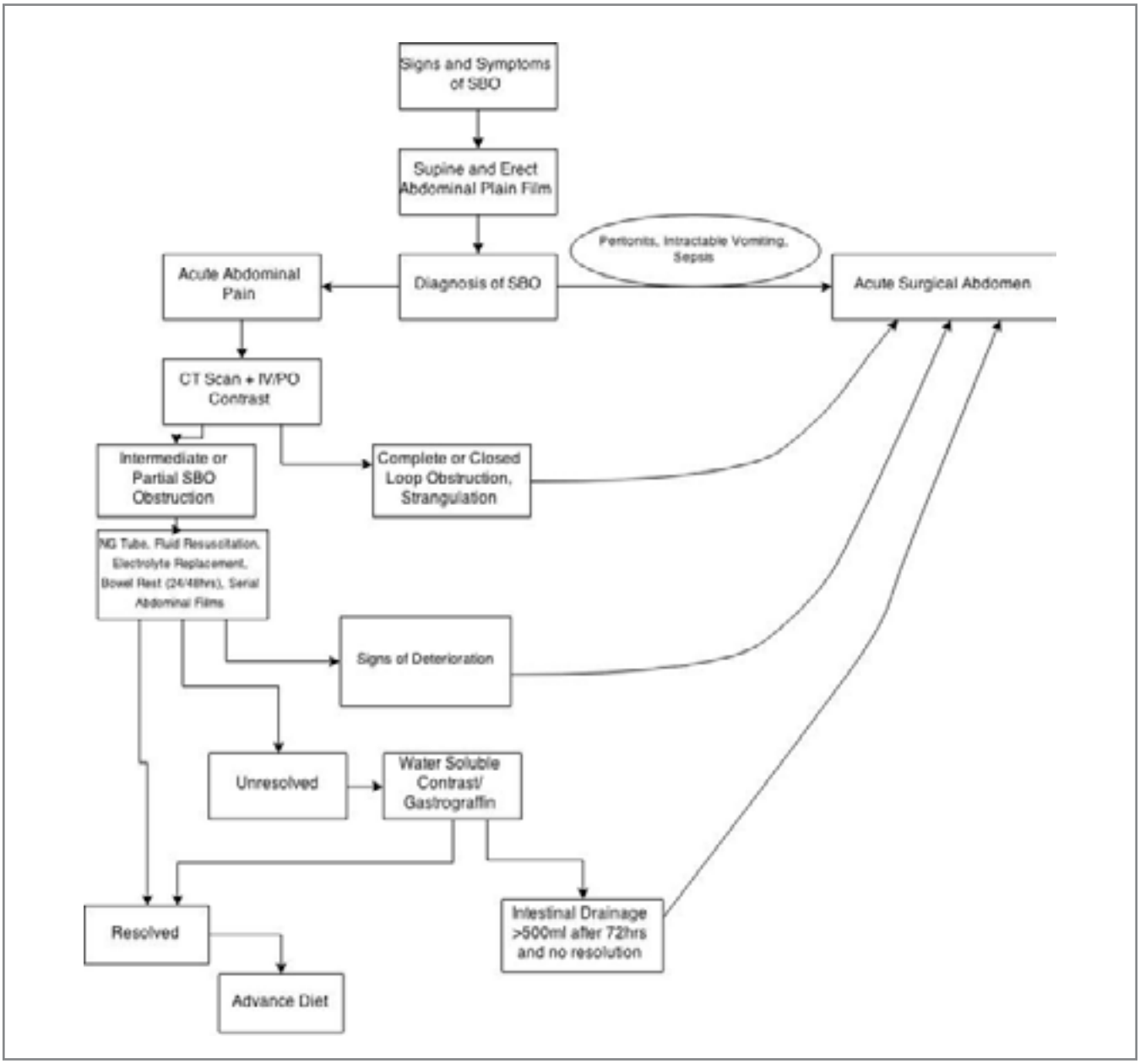
CONSERVATIVE MEDICAL MANAGEMENT VERSUS SURGICAL INTERVENTION

Treatment of AMSBO requires familiarity with conservative medical management and the indications for surgical intervention *(Figure 1, page 24)*. Surgical intervention is controversial because of the potential for inducing adhesions. On the other hand, conservative treatment is often ineffective in eliminating the cause of obstruction. In a study conducted by William et al. showed that patients treated non-operatively have shorter hospital stay (4.9 vs 12.0 days), but higher frequency of recurrence (40.5% recurrence non-operatively vs 26.8% recurrence operatively). They were also found to have a shorter time to re-admission (153 days non-operatively vs 411 operatively).[13] Despite this, initial therapy with conservative management is recommended before surgical intervention in the absence of ominous clinical signs.[4]

Conservative medical management of AMSBO includes bowel rest with early decompression, fluid resuscitation, and correction of electrolyte abnormalities.[14] Early management is important to retard the extent of intravascular volume depletion caused by vomiting and sequestration of volume within the bowel lumen. Factors that lead to this intraluminal

FIGURE 1:

Clinical Management of Mechanical Small Bowel Obstruction



fluid accumulation include decreased reabsorption, increased fluid secretion, associated with rising intraluminal pressure, and a concomitant increase of vasoactive agents, such as prostaglandins and vasoactive intestinal peptide. Blood flow to the bowel may become compromised when intraluminal pressure exceeds intramural capillary and venous pressure.[7] Decompression can be performed using either a naso-gastric tube or a long intestinal tube, neither being more advantageous than the other.[15] Patients without evidence of peritonitis or strangulation can be managed safely with non-operative therapy for up to 72 hours.[4] However, a retrospective study of 123 patients conducted by Cox et al. demonstrated that 31 out of the 38 patients who required surgical intervention did so after 48 hours of conservative management. Most cases of adhesive small bowel obstruction that resolve do so within 48 hours of admission.[16] Persistent ileus or intestinal drainage exceeding 500 mL after 3 days of hospitalization indicate failure of conservative management and warrant the need for surgery.[14] If there is no improvement within 3 to 5 days and surgery is anticipated, parenteral nutrition should be provided and intravenous antibiotics should be initiated.[7]

As mentioned previously, water-soluble contrast medium can be effective in the conservative management of AMSBO. Doses of 50-150 mls, administered either orally or through naso-gastric tube, can be given for up to 48 hours from the time of initiating conservative management.[4] Water soluble contrast medium's mechanism of action is an osmotic shift of fluid into the intestinal lumen which increases the pressure gradient across the site of the obstruction. In general, adverse side effects are rare. Water soluble contrast medium reduces the need for surgery, accelerates the resolution of AMSBO, and shortens hospitalization.[4] However, it does not reduce the likelihood of AMSBO recurrence or obviate the need for surgery with future AMSBO recurrences.[17] Even so, water-soluble contrast medium should be considered in AMSBO patients that warrant conservative management.

The most recognized complication related to the use of water-soluble contrast medium is aspiration pneumonia. This often occurs in patients with delayed gastric emptying or intractable vomiting.[18] Renal failure or anaphylaxis, although rare, can also occur through venous intravasation.[19] Complications can be avoided by ensuring proper placement of the nasogastric tube in the stomach and adequate gastric drainage before administration of water soluble contrast medium.

Indications for immediate surgical intervention include strangulation, peritonitis, intractable vomiting, and complete or closed loop bowel obstruction.[12] Failure to recognize an early indication for surgery may result in serious morbidity or death. For example, strangulation can lead to lethal complications such as endotoxic shock, sepsis, and multiple organ failure accounting for nearly fifty percent of all deaths from small bowel obstruction.[20] Therefore, management of patients with a high index of suspicion for complicated or severe AMSBO requires a low threshold to operate.

Radiographic evaluation can sometimes be helpful in identifying patients that would benefit from immediate surgical intervention. Radiographic evidence of bowel wall thickening, pneumatosis intestinalis, ascites, or mesenteric hematoma can be suggestive of bowel strangulation in the proper clinical context. Closed loop bowel obstruction on CT is characterized by a C or U shaped section of bowel that is dilated along the proximal end but decompressed at the corresponding distal end.[7] Free fluid in the peritoneal space, a frequent finding in patients with AMSBO, could be diagnostic and should be further characterized by volume, density, and location. Focal mesenteric fluid or large amounts of highly attenuated fluid in multiple spaces are suggestive of underlying bowel injury.[21] Mesenteric edema and devascularized bowel may be present in patients with findings of bowel wall thickening, hypoattenuation, bowel dilation, bowel wall hemorrhage, mesenteric fat stranding, or portal venous gas on CT scan.

PREVENTION

There are no current definitive pharmacotherapies or osteopathic manipulative techniques that can prevent the development of mechanical small bowel obstruction in high-risk patients. However, promising results have been demonstrated with the use of statins and sodium hyaluronate carboxymethyl cellulose bioresorbable membrane (Seprafilm) in reducing postoperative adhesion in abdominal surgeries. A retrospective study conducted by S. Srinivasa et al. demonstrated that patients who presented with adhesive small bowel obstruction and were on a statin had a decreased need for therapeutic surgery in order to relieve the obstruction.[22] Statins have been shown to increase peritoneal fibrinolytic activity, decrease post-surgical inflammatory response, and reduce intestinal ischemia and reperfusion injury, all of which are risk factors associated with ASBO and subsequent operative management. Seprafilm membranes have been demonstrated in numerous studies to reduce the risk of abdominal adhesion following abdominal surgery.[23] These therapeutic measures are most beneficial following surgery and are often out of the hands of a primary care physician. Therefore, the best preventative measure a primary care physician can implement is to properly manage the underlying disease associated with the increased risk, such as Crohn's Disease. Dietary modifications have been shown to decrease the risk of developing complete obstruction in such patients. Dietary modifications should be implemented to avoid fiber-rich foods that increase gas and bulk within the bowel.[24] PCPs should advice such patients to eat vegetables that are cooked, canned, and free of skin and seeds. Avoidance of fatty and greasy foods as well as lactose containing drinks should also be advised.

CONCLUSION

Acute mechanical small bowel obstruction is a common emergency and can present initially in the outpatient setting. PCPs should consider pertinent medical history and physical exam findings when conducting a clinical evaluation of AMSBO. All patients with suspected AMSBO should be hospitalized and treated initially with conservative management. This includes bowel rest with early decompression, fluid resuscitation, and correction of electrolyte abnormalities. Water-soluble contrast medium can be useful adjunct in this approach; it has both diagnostic and therapeutic purposes. Furthermore, water-soluble contrast medium is safe and reduces the need for surgery, time to resolution and hospital stay. Non-operative management can be provided safely, in the absence of strangulation or peritonitis, for up to 72 hours. In contrast, ambulatory patients presenting with ominous clinical signs and symptoms should be considered for immediate surgical intervention. Indications for surgery include strangulation,

peritonitis, intractable vomiting, complete or closed loop bowel obstruction, or failure to improve after 72 hours of conservative management.

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REVIEW ARTICLE

Dysuria: A Focus on Urinary Tract Infections

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KEYWORDS:
Urinary Tract Infection
Cystitis
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Pyelonephritis
Recurrent UTI

Urinary tract infections are one of the most common infections faced by the primary care physician with nearly half of all women being diagnosed during their lifetime. Although easily treated with anti-microbial agents, urinary tract infections typically recur or are often incompletely resolved. New strategies are being developed for both treatment and prophylaxis of the disease process. In this review, the epidemiology, pathogenesis, treatment recommendations, and emerging research surrounding urinary tract infections will be discussed.

INTRODUCTION

Urinary tract infections (UTIs) are the most commonly encountered bacterial infection in the outpatient setting and are estimated to account for over eight million infections in the United States annually.[1] The majority of patients diagnosed with an acute UTI are prescribed antibiotics and 1% requires hospitalization.[2] This in turn results in over two billion dollars being spent every year for evaluation and treatment.[3] Therefore, a true understanding in the management and evaluation of these infections is vital for the primary care physician. This review will discuss the epidemiology, pathogenesis, treatment recommendations, and emerging research surrounding UTIs.

EPIDEMIOLOGY

Infections involving any section of the urinary tract are considered to be urinary tract infections (UTI). This includes urethritis, cystitis, ureteritis, and pyelonephritis.[2] In 2007, there were 10.5 million ambulatory visits for UTI with one fifth of these visits being to the emergency department.[4] UTIs are the most common primary diagnosis in women who present to the emergency department with more than 50% of having at least one UTI in their lifetime, and 11% of women being diagnosed with a UTI every year.[1, 5, 6] The higher incidence of UTIs in women can be related to the anatomy of the lower genital urinary tract. The shorter urethra in women in conjunction with colonization of the vaginal introitus with gastrointestinal pathogens can place women at higher risk of UTIs as compared to men.[7, 8]

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While numerous risk factors have been defined for the development of UTIs, the majority of UTIs occur in individuals without any of the defined risk factors. The greatest risk populations are sexually active women aged 20-40 years and postmenopausal women over the age of 60 years.[9] The incidence of UTI in young sexually active women ranges between 0.5 to 0.7 UTIs per person year and by the age of 24 one third of women will have been diagnosed with a UTI.[10, 11]

DIAGNOSIS

UTIs are often labeled as either uncomplicated or complicated. Uncomplicated UTIs are defined as a symptomatic bladder infection with laboratory tests consistent with a UTI; while a complicated UTI is defined as a symptomatic UTI caused by functional or structural abnormality, having had urinary instrumentation, having systemic diseases such as renal insufficiency, diabetes, or immunodeficiency, or having undergone organ transplantation.[12] UTIs can also be broken down into three separate categories: cystitis, asymptomatic bacteriuria and acute pyelonephritis.

CYSTITIS

Cystitis is commonly seen in the primary care office and is characterized by dysuria, frequency and urgency of urination with or without associated suprapubic pain in premenopausal women but malaise, nocturia, incontinence and foul smelling urine in post-menopausal women.[13, 14] Although cystitis produces significant short-term morbidity, there are little to no long-term consequences.[13] In non-pregnant women, these infections have no long-term adverse effects on renal function, no increased mortality, and if left untreated rarely

progress to upper urinary tract infections.[15] This holds true even for individuals with recurrent cystitis.[15] When treating uncomplicated cystitis with antibiotics, women are more likely to have complete clinical symptom resolution and microbiological eradication.[16] However, they also have higher rate of adverse events secondary to the usage of antibiotics.[16]

The diagnosis of a UTI should first be based on the patient's symptoms but should also be used in conjunction with a urinalysis. Urinalysis with presence of red blood cells, high nitrite levels, and leukocyte esterase are concerning for a potential underlying UTI.[2] A positive urinalysis should be followed by urine culture with anti-microbial susceptibility testing, which is the gold standard for a definitive diagnosis.[2] Urine culture allows for identification of the causative agent and for antimicrobial susceptibility testing to be performed. Antibiotics should then be tailored accordingly to the results. Previously $\geq 10^5$ colony-forming units (CFU)/mL of midstream urine was considered a positive culture, however, recent literature has changed the diagnostic criteria to include 10^3 CFU/mL urine, in the presence of overt UTI symptoms.[2,17] Following treatment, repeat urinalysis for cure is currently not recommended unless symptoms recur.[18]

ASYMPTOMATIC BACTERIURIA (ASB)

Asymptomatic bacteriuria (ASB) is defined as bacteria in the urine without clinical signs or symptoms of a UTI.[9] The overall prevalence of ASB in women is 3.5% and increases following sexual intercourse.[19] In women aged 5-14, 1% will have ASB with the rate of this condition increasing with age.[20] When over age 80, 20% of women will have a diagnosis of ASB.[20] The prevalence of ASB in pregnancy ranges from 2 to 20% with one fifth of these patients developing acute pyelonephritis if left untreated.[21]

ASB is a microbiologic diagnosis that occurs when the detected organism outcompetes a uropathogenic organism.[9] This in turn prevents a clinical manifestation of infection. Therefore, routine screening and treatment for ASB is not recommended in the general population except during pregnancy.[6,9] Screening and treatment of ASB during pregnancy could decrease maternal and fetal mortality by 77%.[22] In the general population, however, treatment of ASB can lead to development of a UTI and antimicrobial resistant bacteria.[23]

ACUTE PYELONEPHRITIS

Acute pyelonephritis is defined by an infection of the renal pelvis and kidney that usually results from ascent of a bacterial pathogen up the ureters from the bladder to the kidneys.[24] The incidence of acute pyelonephritis is 59.0/10,000 for females and 12.6/10,000 for males in the general population with a hospitalization rate of 11.7/10,000 for women and 2.4/10,000 for men.[25,26] A good history and physical examination is the key to aid the physician in the diagnosis. Patients may present with or without urinary symptoms. Associated symptoms for acute pyelonephritis include fever, chills, back/flank pain, nausea or vomiting.[6,24] On physical examination, costovertebral angle tenderness is almost universal to pyelonephritis and its absence should raise the question of an alternate diagnosis.[24]

Diagnostic testing should include urinalysis and urine culture with susceptibility. The presence of white blood cell casts indicates a renal origin of pyuria and is highly indicative of pyelonephritis.[24] Post-treatment urinalysis and culture are not warranted in asymptomatic patients but may be of value in those whose symptoms do not improve in three days.[24] Imaging is not warranted in those who remain asymptomatic but those who have recurrence of symptoms or do not respond to therapy in 72 hours should undergo imaging to assess for a structural abnormality.[27] According to the American College of Radiology CT of the abdomen and pelvis is the diagnostic imaging modality of choice.[27]

PATHOGENESIS

The most common pathogen associated with UTIs is Escherichia coli (*E. coli*), an opportunistic pathogen.[2,6,9] When examining community-acquired UTIs, *E. coli* is the source in 80-90% of all cases and constitutes 74.4% of cases in the outpatient setting, 65% of hospital-acquired infections and 47% of health care associated infections.[9,28] Various *E. coli* strains have been shown to be commonly shared among pets, humans (non-sex and sex partners), and between humans and pets.[29,30] It is therefore not uncommon for multiple individuals within the same household to be infected with the same strain of *E. coli*.

UTIs are felt to develop via the fecal-perineal-urethral route of infection since most uropathogens, including *E. coli*, originate in the rectal flora.[9] The uropathogens then have an interim phase of vaginal and periurethral colonization and subsequently enter the bladder via the urethra.[31] It is unclear the exact means of ascension through the urinary tact but it is widely felt that motility mediated by flagella may play an important role in *E. coli* infections.[32]

The first line of defense for UTI prevention is the hosts response to inhibit attachment of *E. coli* to the urothelim. This is completed via the flow, acidity, and the high osmolarity of urine.[33] *E. coli* that avoid the first line of defense are able to invade the urothelium and then activate a release of immune mediators including cathelicidin or defensins and interleukin 6 and 8.33. Following the release of immune mediators, type 1 fimbrial adhesion FimH binds to the integrin of the bladder urothelium.[34] A signal cascade is activated that allows the

E. coli to be surrounded and enveloped by the host plasma membrane.[34] Once the *E. coli* are brought into the membrane, they rapidly multiply and form intracellular bacterial communities that exhibit biofilm like properties.[35] The intracellular bacterial communities are then able to dissociate and migrate out of the urothelium and then reinvade the urothelium, which reinitiates the entire process.[36]

Although *E. coli* is the most common pathogen to cause UTIs, several other pathogens do cause UTIs in varying degrees. The gram negative organisms *Klebsiella* spp, *Pseudomonas aeruginsoa* and *Proteus spp* and the gram positive organisms *Streptococcus agalactiae* and *Staphylococcus saprophyticus* all can cause UTIs in the general population.

RECURRENCE

Recurrent UTIs are defined as two uncomplicated infections within six months or three infections within one year with at least one confirmed with a urine culture.[1] The probability for recurrence following an initial UTI is 25% at six months and 46% at one year for young women.[2, 37] Also a patient's likelihood of recurrence increases if they have had more than one prior UTI.[37] If close temporal proximity between infections is encountered, the etiology of the infections are more likely to be caused by the same strain of bacteria.[37] Persistence and relapse of UTIs are most common with phylogenetic group B2 *E. coli* while cure and reinfection UTIs are common with the phylogenetic group D E. coli. [9]

Recurrent UTIs have multiple possible etiologies. The same uropathogenic strain can persist in the gastrointestinal tract and can repeatedly colonize the bladder.[37] When relapse with the preceding infecting *E. coli* occurs, the strains are felt to have a greater biofilm formation capacity which allows better adherence to the bladder. Alternatively, a different uropathaogenic strain can be introduced into the gastrointestinal tract or a new strain can be directly introduced from the environment into the perineal area and invade the bladder.[37]

A final etiology for recurrent UTIs is quiescent intracellular reservoirs. Quiescent intracellular reservoirs remain in the bladder urothelium following clearance of the initial UTI.[9] These remain undetected by the immune system and are less susceptible to antimicrobials. *E. coli* is therefore able to persist inactive for prolonged periods of time within the bladder urothelium.[9, 37] Stimulation of facet cell exfoliation leads to activation of the quiescent intracellular reservoirs.[9] This then causes cell differentiation and proliferation cascades which results in recurrence of infection.[9] Low levels of estrogen have been shown to increase the levels of quiescent intracellular reservoirs.[9] This serves as an explanation for recurrent UTIs in postemenopausal women.

TREATMENT

Following diagnosis of a UTI, spontaneous resolution of symptoms and sterilization of the urine occurs in approximately 25% of patients after 5-7 weeks with no antibiotic treatment and this number rises to 80% at 5 months.[38] However, it has been shown that antimicrobials are superior to placebo in clinical and microbiological success due to the timeliness of eradication.[39] After anti-microbial initiation, patients should begin to note symptom relief by 36 hours.[40]

Currently no single agent is considered the best agent for uncomplicated cystitis according to the Infectious Diseases Society of America and the European Society for Microbiology and Infectious Diseases latest recommendations.[41] Empiric treatment of uncomplicated cystitis should be based upon on the susceptibility patterns in the physician's area, risk of adverse effects, resistance rates and the risk of collateral damage.[41] Nitrofurantoin or fosfomycin trometamol were both given the highest recommendation for treatment of uncomplicated UTIs and if *E. coli* resistance rates are known to be less than 20%, trimothroprim-sulfamethoxazole is also an acceptable first line agent.[41] The fluoroquinolones also carry a high recommendation for treatment of uncomplicated UTIs in short duration but should be considered an alternative secondary to their propensity for collateral damage.[41]

Beta lactam agents including amoxicillin-clavulanate, cefdinir, cefaclor, cephalexin and cefpodoxime-proxetil are considered appropriate choices for therapy if other agents cannot be used.[41] However, the beta lactam agents have a propensity for decreased efficacy and increased adverse effects as compared to other anti-microbials.[41] Additionally, ampicillin and amoxicillin/clavulanic acid are not recommended due to high resistance patterns worldwide.[41]

For acute uncomplicated pyelonephritis both ciprofloxacin and trimothroprim-sulfamethoxazole should be considered first line agents if the relative resistance rate of the uropathogens is low *(Table 1, page 30)*.[41] However, if the resistance rate is unknown for trimothroprim-sulfamethoxazole or greater than 10% for the fluoroquinolone then a single intravenous does of a long acting anti-microbial is recommended prior to starting either of these.[41] Oral beta lactam agents are currently less effective than other anti-microbials for acute pyelonephritis and if used should be prescribed for between ten and fourteen days.[41]

REDUCTION OF UTIs

Prophylaxis with antimicrobial agents can be an effective strategy to reduce the incidence of UTIs but does come with possible adverse effects. Low-dose daily antibiotic prophylaxis in the means of nitrofurantoin , cephalexin, or trimethoprim-sulfamethoxazole has been shown to reduce the incidence of

TABLE 1:
Treatment of uncomplicated UTIs in the non-pregnant population

	Dose	Dose	Duration
Asymptomatic Bacteriuria	No treatment recommended		
Cystitis	First Line		
	Fosfomycin	3 grams	Single Dose
	Nitrofurantoin	100 mg BID	Five Days
	Trimethoprim/sulfamethoxazole	160/800 mg BID	Three Days
	Second Line		
	Ciprofloxacin	250 mg BID	Three Days
	Ciprofloxacin, extended release	500 mg QD	Three Days
	Levofloxacin	250 mg QD	Three Days
	Ofloxacin	200 mg QD	Three Days
	Third Line		
	Amoxicillin/clavulanate	500/125 mg BID	Seven Days
	Cefdinir	300 mg BID	Ten Days
	Cefpodoxime	100 mg BID	Seven Days
Pyelonephritis	Ciprofloxacin	500 mg BID	Seven Days
	Ciprofloxacin, extended release	1000 mg QD	Seven Days
	Levofloxacin	750 mg QD	Five Days

UTIs.[42] If a patient's UTIs are known to coincide with sexual activity, a single dose of antibiotic administered postcoitally is recommended.[43]

Caution must be used with routine use of antibiotic prophylaxis. Daily administration of antibiotics increases the antimicrobial resistance in both the gastrointestinal and urinary tracts. After one month of daily prophylaxis with trimethoprim-sulfamethoxazole, 86% of fecal *E. coli* and 91% of bacteriuria *E. coli* display resistance.[44] While nitrofurantoin has a significantly lower risk of resistance, it is also less effective against uropathogens aside from *E. coli* and is not recommended for long-term use in elderly patients or those with decreased renal function.[45-47] Furthermore, the presence of quiescent intracellular reservoirs may enable bacteria to be entirely intractable to any antimicrobial treatment and prophylaxis.[48]

Many alternative strategies to reduce the risk of recurrent UTIs are known and even more are being developed. These include modification of external risk factors including diaphragm or spermicide use, patient-initiated antibiotic treatment at an early stage, immunoactive prophylaxis, prophylaxis with food additives, and local hormone treatment in postmenopausal women.[16] Cranberry products have extensively been studied regarding the efficacy in UTI prevention. These products have been shown to decrease the ability of *E. coli* to adhere to the urothelium and also within human vaginal epithelial cells.[49,50] However, two recent meta-analyses showed that cranberry only carries a slight protective effect against UTI in the general population.[51, 52] Further investigation and research is necessary to determine if these products are able to effectively treat or prevent UTIs.

Methenamine salts have also been used in the prophylaxis of UTIs.[53] A recent Cochrane Review reported that methenamine may be effective for preventing UTIs in those without renal tract abnormalities or a neurogenic bladder in the short term.[55] However, it is recommended that further randomized controlled trials need to be conducted in order to assess the efficacy of methenamine's long term potential.[55]

A pilot study comparing the efficacy of ciprofloxacin versus ibuprofen for the treatment of uncomplicated UTIs was

recently completed. It was shown that ibuprofen has potential utility for this indication. Both treatments showed equal efficacy for symptom resolution and bacterial clearance with ibuprofen being non-inferior to ciprofloxacin. Though this study was underpowered, it suggests that the anti-inflammatory effect may help to eradicate uropathogens but further validation is needed.[54]

A vaccine to protect against urinary tract infections is also being developed. It consists of a vaginal suppository containing ten heat-killed strains of uropathogenic bacteria.[55] A total of six strains of *E. coli* and one strain each of P. mirabilis, M. morganii, K. pneumonia, and E. faecalis are included.[55] During phase 2 testing the vaccine has been shown to reduce the incidence of *E. coli* UTI in sexually active women aged 20-50 with a history of recurrent UTIs.[55] However, no statistically significant difference was seen in development of anti–*E. coli* antibody levels between the experimental and placebo group.[55] Further investigation through a phase 3 trial is required prior to routine utilization.

CONCLUSION

In conclusion, UTIs remain a large burden on society despite the ease in which they can be treated. Recurrence rates are typically high as compared to other disease processes despite adequate anti-microbial treatment. The primary care physician should be aware of current recommendations for both treatment and prophylaxis of UTIs in the general population. Further research is needed to determine strategies to better treat not only the disease but also to halt its recurrence.

DISCLOSURES:

Dr. Ashurst – Author for Emergency Medicine Practice with the company EB Practice, LLC.

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REVIEW ARTICLE

Acute Lower Urinary Tract Infection Caused by Lactococcus Garvieae

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KEYWORDS:

Urinary Tract Infection

Lactococcus Garvieae

We report the 19th case of human infection by Lacotcoccus garvieae and only the 2nd case causing a urinary tract infection. This organism may be an emerging zoonotic pathogen in immunocomprimised individuals.

INTRODUCTION

The *Lactococcus* genus was separated from the Streptococcus genus in 1985 on the basis on genetic analysis. *Lactococcus garvieae* human infections are rare.[1] This unusual pathogen is considered to be of low virulence in human beings except for those with an immunocompromised state.

Lactococcus garvieae is primarily a fish pathogen affecting salt water fish of the Far East (Japan, China). The same organism has been isolated from mastitis infections in cows and water buffalos. Human infection is presumed to be primarily through contaminated cows milk, cheese, or raw fish products. Consumption of raw fish during the summer months and fish handlers who manipulate raw fish have long been suspected as the most probable sources of infections in humans. Risk factors include anatomically or physiologically altered gastrointestinal tract, long term use of H2 blockers or proton-pump inhibitors (PPI's),valvular heart disease and any immunocomprimising condition, such as cirrhosis, chemotherapy, cancer, autoimmune disease.

Human infections total eighteen cases in the English literature in 2014. We report a nineteenth case. The majority of the previously reported cases are infective endocarditis. There have been isolated cases of acalcular cholecystitis, spondylodiscitis, primary septicemia, hip prosthesis infection, liver abscess, peritonitis, diverticulitis and urinary tract infection. [1, 2, 3, 4, 5, 6, 7] We report the second case of a urinary tract infection with *Lactococcus garvieae* in a young female. The previously reported case of a urinary tract infection occurred in an elderly male who underwent a transurethral prostatic resection (TURP). *Lactococcus garvieae* was isolated from both urine and blood cultures in this patient.[1]

CASE HISTORY

A thirty-six year old Caucasian female was admitted through the emergency department with a two day history of nausea, vomiting & suprapubic abdominal pain. She reported mild dysuria and polyuria. Original urinalysis demonstrated 10-20 WBC's / HPF, 2-4 hyaline casts, and 2+ bacteria. Serum vitamin B12 levels were low at 167pG/ml (180-194), low serum iron 24 mcg/dL (40-135) and a positive urine drug screen for benzodiazepines and cannabinoids. Urine culture revealed *Lactococcus garvieae* as the sole microbiologic agent. Blood cultures were negative for growth.

Two months prior to this admission the patient was hospitalized at a different facility for thrombotic thrombocytopenic purpura (TTP) and Staphylococcus aureus sepsis of an unknown source. This patient has long history of ETOH abuse and dependency associated with recreational drug use. She also has a history of chronic recurrent pancreatitis. She received oral B12 supplementation, thiamine and ceftriaxone one gram IV for 72 hours and was discharged on oral amoxicillin.

DISCUSSION

The *Lactococcus genus* is composed of 8 species and subspecies. *Lactococcus garvieae* is by the far the most common etiologic agent associated with human disease.[1] Human infections with *Lactococcus garvieae* are rare and generally associated with an immunocompromised host. Human infections are associated with significant morbidity and up to a 25% mortality rate.[2]

Lactococcus garvieae is uniformly resistant to clindamycin. Therapy should consist of beta-lactam antibiobitics with or without aminoglycosides or ciprofloxin. Septicemic or endocarditis patient will require prolonged intravenous antibiotic therapy.[1]

The port of entry for *Lactococcus garvieae* has been suggested to a gastrointestinal defect such as a gastric ulcer, inflamed diverticulum, prior GI tract surgery, long term use of

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H2blockers or PPI's. Generally no source of entry can be identified. However, manipulation of contaminated fish with unprotected skin surfaces may increase the risk of bacteremia.[3,8]

We report a second case of *Lactococcus garvieae* urinary tract infection. Our patient was without urinary tract manipulation or surgery. Our patient denied exposure to raw fish or raw consumption or any skin lesions that could act as a vehicle for a source of entry. Her risks factors include intermittent long term PPI therapy, ETOH abuse, recreational drug use, TPP. We are unsure if a serum cyanocobalamin deficiency or serum iron deficiency may predispose one to a UTI with this organism. It is unclear how this patient acquired a UTI with this emerging zoonotic pathogen.

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REVIEW ARTICLE

Radiotherapy Induced Tissue Injury: Mechanisms, Symptoms & Management

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KEYWORDS:

Radiation Therapy

Previous literature reviews in the field of cancer and radiation research have focused primarily on the molecular and pathological findings observed in cancer patients receiving radiotherapy. This article goes a step further to provide the primary care physician a systematic review of the signs and symptoms associated with the adverse effects of radiation therapy and their implications for treatment and long-term prognosis. A review of relevant literature was conducted for articles published within the past 10 years and of these, 21 were included in this review. From a primary care standpoint, this study focused strongly on clinically relevant side effects as a result of radiation therapy in the gastrointestinal, pulmonary, cardiac, and dermatologic systems. Thus, by providing clinical knowledge regarding the treatment and management of these patients, physicians may improve quality of life and overall survival.

INTRODUCTION

Cancer is the second leading cause of death in the United States. It is estimated that more than 1.6 million new cases of cancer will be diagnosed in 2014, and of these, almost two-thirds will be treated with radiation therapy.[1] While the benefits of using radiation therapy in the treatment of cancer have been well established, the development of acute and chronic adverse effects and their implications on long-term morbidity and mortality remain largely unknown. Conversely, recent advances in radiation oncology have led to significant improvements in patient outcomes by adjusting therapy to patient specific factors including tumor size, normal tissue radiosensitivity, and radiation dosage.[2] As such, the current goals of mainstream radiation therapy are to maximize tumor reduction and minimize radiation-induced adverse effects.[3] Clinical knowledge of the signs and symptoms most frequently associated with radiation-induced toxicity is vital in cancer survival due to the fact that radiation toxicity may limit the use of some treatment modalities. It has been well established that toxicity-related interruptions in radiation therapy are associated with decreased patient survival.[4] While acute radiation toxicity tends to be self-limiting, late-onset toxicity may have a severe impact on quality of life many years after radiation exposure. In addition to the use of therapeutic interventions to reduce side effects, careful management of disease progression is an important factor in improving quality of life and prognosis.[3] The aim of this article is to provide the primary care physician a systematic review of signs and symptoms associated with the side effects of radiation therapy and their implications for current treatment modalities and long-term prognosis. For a comprehensive review regarding the underlying pathology and diagnostic studies related to radiation-induced damage, the reader is encouraged to refer to additional outside articles.

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MATERIALS & METHODS

A systematic review of the literature was done from 2004 to 2014 by use of PubMed and Medline databases. The search terms included radiation therapy, acute and late toxicity, pathophysiology, management, gastrointestinal, radiation pneumonitis, cardiac toxicity, and radiation dermatitis. References were screened and selected for inclusion in this review based on relevance and of these, 21 were included in this review.

GASTROINTESTINAL TOXICITY

It is estimated that over 200,000 patients a year undergo radiation therapy for pelvic and gastrointestinal cancer.[4] Of the patients treated with pelvic radiation therapy, 60-80% will experience acute bowel toxicity, as well as significant long-term impacts on their quality of life.[4] Further, as many as 50% of patients receiving pelvic radiation therapy experience chronic GI dysfunction or changes in bowel habits.[4,9] Considering these findings, clinically significant adverse effects and radiation toxicity remain a health concern.[4] It has been shown in epidemiological studies that smoking and previous metabolic syndromes, such as hypertension, diabetes, and inflammatory bowel disease, increase the risk of acute and chronic radiation enteropathy.[9] Psychosocial factors

involved in cancer diagnosis and treatment have also been implicated in GI dysfunction.[9] In addition, the intestines are more susceptible to radiation exposure during pelvic radiation therapy due to their large surface area. Surprisingly, physicians investigate complaints of gastrointestinal issues made by patients receiving radiation therapy less frequently than those not receiving radiation therapy.[4] Thus, the identification and management of new onset symptoms are key in modifying therapeutic and long-term outcomes.

Acute bowel toxicity presents within the first couple of weeks of radiation therapy as diarrhea, nausea, and abdominal pain. These symptoms typically subside 3-4 weeks after the conclusion of therapy.[4] In general, the treatment of acute radiation enteropathy is primarily symptomatic. It is important to mention that severe complications, although only thought to occur in 4-8% of patients 5-10 years after treatment, are life threatening and include fistulation, sepsis, stenosis, gastrointestinal failure, and secondary malignancies.[10] These life threatening complications are treated as emergencies and surgical intervention is indicated.[10] The exact mechanisms resulting in acute radiation toxicity have not yet been fully elucidated; however, epithelial injury has been associated with degeneration of the mucosal layer and the release of inflammatory mediators.[4]

Symptoms of chronic bowel toxicity typically occur between six months and three years following radiotherapy and include diarrhea, rectal bleeding, abdominal cramping, constipation, and changing bowel habits.[9] Similarly, the exact mechanisms involved in chronic toxicity remain unclear. Management of symptoms is focused primarily on the treatment of the underlying functional deficits.[9] This implication highlights the importance of recognizing the underlying pathology as it determines the appropriate treatment modality. For example, radiotherapy-related diarrhea is frequently reported following pelvic radiation therapy and has been associated with physiological changes in bowel motility, small-bowel bacterial overgrowth, and malabsorption of bile salts and carbohydrates.[4] Awareness of the symptoms associated with radiation-induced bowel toxicity and the appropriate diagnostic studies are fundamental in the management of an increasing population receiving radiation therapy. In addition, a better understanding of the pathological process of gastrointestinal toxicity is necessary to identify possible therapeutic interventions in the goal of preventing interruptions in cancer therapy and improving patient quality of life.

PULMONARY TOXICITY

Lung and bronchus cancer are the leading causes of cancer related death in the United States. It is estimated that 224,000 new cases of lung and bronchus cancer will be diagnosed in 2014 and continues to be a global health burden.[1] Thoracic radiation therapy is an essential treatment modality in the treatment of lung cancer, as well as a variety of other thoracic tumors. Unfortunately, clinically significant tissue toxicity is a frequent dose-limiting adverse effect in patients receiving radiation therapy, consequently reducing the effectiveness of therapy.[11] Radiation pneumonitis (RP) and pulmonary fibrosis (PF) are the most common severe adverse effects in these patients and represent a significant barrier to patient outcomes and overall survival rates.[11] It has been reported that between 5-50% and 1-43% of patients undergoing thoracic irradiation experience radiation pneumonitis and pulmonary fibrosis, respectively.[12,13] Further, a number of patient-related factors have been linked to the development of RP, such as prior lung disease, reduced pulmonary function, genetic factors, and old age. In addition, the radiation dose rate, absorbed radiation dose, and the volume of lung irradiated are factors that determine the level of risk of developing radiation-induced lung injury (RILI).[11] However, a general consensus has not yet been established regarding lifestyle and comorbidity risk factors due to the frequent contradictions between studies.[12]

The exact mechanism of radiation-induced lung injury is complex and has not been described in detail; however, it is suggested that tissue repair and cellular signaling pathways associated with chronic inflammation are involved and include inflammatory mediators, immune cell recruitment, and macrophage activation among others.[11] For example, TGF-β, a cytokine produced by inflammatory cells after radiation-induced tissue damage, is involved in a wide range of cellular signaling pathways implicated in RILI, such as inducing the differentiation of fibroblasts into matrix-producing myofibroblast, inhibiting epithelial cell proliferation, and controlling the breakdown of connective tissue.[11] The pathological changes associated with RILI are described in a three-step process that involves a latent phase, exudative phase, and fibrotic phase.[3] The latent phase is described as occurring up to three months after radiation therapy, yet no significant histological findings are typically observed. During the exudative phase, acute inflammation is observed, as well as inflammatory cell infiltration and thickening of the interstitium. Consequently, alveolar gas exchange is reduced. Finally, the fibrotic stage is characterized by permanent fibrosis, which contributes to the reduction in the number of alveoli, thickening of alveolar septae, and thickening of the alveolar wall.[11, 12] The alveolar-capillary complex is the most radiosensitive component of the lung and is highly resistant to treatment.[11]

RP represents the acute phase of radiation-induced lung injury (RILI) and typically occurs 1-6 months after radiation therapy.[11] Clinical manifestations of RP typically appear 1-3

months following radiation therapy and include a dry cough, dyspnea, fever, respiratory insufficiency, and chest pain.[11, 12] While there is little evidence supporting the effectiveness of therapeutic intervention, symptoms are frequently managed with oral corticosteroids.[14] The onset of radiation-induced pulmonary fibrosis, a chronic phase of RILI, typically occurs months to years after treatment.[12] Progressive pulmonary fibrosis by this stage is usually permanent and may lead to late complications that include cor pulmonale and respiratory insufficiency.[13] In addition, both the acute and late toxic events reduce the capacity to recover from ongoing or future pulmonary challenges and in some cases, may be fatal.[12] The current approach in the treatment of RILI is focused on balancing the inflammatory component of lung injury. For example, amifostine, a free radical scavenger, is a therapeutic agent currently in clinical use to reduce the oxidative stress associated with radiation exposure.[11] In addition, the neutrophil elastase inhibitor, sivelestat, has been shown to significantly reduce collagen deposition and prevent fibrotic changes associated with RILI.[11] While a few pharmacological agents are in current clinical use, further research into the effectiveness of existing and future therapeutic agents is necessary. It is also important to recognize that clinical factors, such as concurrent chemotherapy, re-irradiation, and the recent withdrawal of steroids are associated with an increased risk of RP and may require more extensive monitoring.[13]

Currently, imaging studies are the mainstream tool for evaluating pulmonary toxicity. MRI imaging has largely replaced the use of commuted tomography (CT) and X-rays in order to avoid further radiation exposure in radiation-susceptible patients.[11] Radiographic findings of RILI show areas of infiltration and scar formation near or around the site of radiation exposure. In addition, pulmonary function testing, forced vital capacity, and carbon monoxide diffusion capacity are also utilized clinically as a measure of lung injury.[12] Despite a number of diagnostic modalities and scoring systems, RP is largely diagnosed based on the clinical symptoms following radiation therapy. Current research is focused on the association between specific serum biomarkers and RILI to provide better diagnostic measures for tissue damage. Due to the fact that cytokines are associated with the fibrotic and inflammatory changes associated with RP, these biomarkers offer a potential mechanism to monitor tissue toxicity during radiation therapy. Studies have shown that elevated plasma levels of both TGF-β and IL-6 are associated with an increased risk of RP.[3, 11]

Thoracic radiation therapy exposes other critical organs to radiation damage, such as the heart, trachea, bronchus, and esophagus. Although, the trachea and bronchus are at increased risk for radiation exposure, they appear to be relatively radioresistant.[3] Nevertheless, radiation esophagitis, a common adverse effect following thoracic radiotherapy, remains a dose-limiting complication in the treatment of lung cancer. Research estimates that as many as 30% of patients undergoing pulmonary chemoradiation experience radiation esophagitis, most commonly complaining of dysphagia.[3] Progression of this disease may lead to severe complications, such as esophageal stricture and ulceration, which may require immediate hospitalization and surgical intervention.[3, 15] Thus, complications of radiation esophagitis have implications on quality of life and ultimately, overall survival. Monitoring and managing of these symptoms is fundamental to enhance patient outcomes. In addition, the utilization of modern radiation techniques that involve risk planning, avoidance of at risk organs, and planned dose/volume irradiation are essential in reducing esophageal toxicity.[3,15] Therapeutic interventions for radiation esophagitis are primarily aimed at symptomatic relief using agents such as topical analgesics; however, current research on the effectiveness of radioprotective agents is ongoing and may provide additional strategies for the prevention of radiation damage.[3]

CARDIAC TOXICITY

Thoracic radiation therapy is indicated in the treatment of a number of thoracic malignancies, such as Hodgkin' lymphoma, lung cancer, breast cancer, and other mediastinal cancers.16 Although vast improvements in radiation techniques over the past few decades have increased cancer survival rates and reduced radiation-induced adverse effects, cardiovascular disease remains one of the most severe and life-threatening complications, carrying clinically significant morbidity and mortality.[8] Radiation-induced heart disease (RIHD) is considered a disease of long-term cancer survivors and is the leading cause of non-malignant mortality in these patients.[17] As such, little data is currently available regarding the long-term benefits of modern tissue-sparing radiation techniques on reducing RIHD. In fact, cancer survivors who underwent radiation therapy as a child are at increased risk for developing late cardiac complications. It is estimated that cardiovascular complications manifest within 3 to 29 years after completion of radiation treatment with an incidence between 10% and 30% by 5 to 10 years.[11,13] Lifestyle factors, such as prior cardiovascular disease, obesity, young age, diabetes, hypertension, and smoking further compound cardiac risk. Systemic chemotherapy has also been recognized as having a synergistic effect with concurrent radiotherapy in the development of cardiovascular disease.[16] In addition, several studies have shown a significant increase in cardiovascular-related mortalities in patients receiving left-sided radiotherapy as opposed to right-sided radiotherapy.[16] Management of these risk factors and the use of routine screening protocols are crucial in preventing morbidity and mortality. While there are no uniform guidelines for screening and monitoring post-

irradiation cardiac damage, a baseline evaluation of cardiac function is recommended to monitor disease progression.[17]

The features of RIHD are complex and are further compounded by the specific underlying malignancy. Acute and chronic cardiac adverse effects of thoracic radiation therapy include pericarditis, coronary artery disease, valvular dysfunction, conduction system disruption, and heart failure.[17] In general, acute RIHD must be considered in any patient presenting with cardiovascular complaints at the time of radiation treatment; however, these complications are not usually clinically significant.[16] For example, acute pericarditis, a now uncommon complication due to advancements in radiation techniques, may present similarly to traditional forms of acute pericarditis with symptoms of fever, pleuritic chest pain, dyspnea, and tachycardia.[16] Acute pericarditis is not considered a dose limiting complication and is frequently resolved with bed rest and NSAIDS.[18] Conversely, as many as 10% to 20% of patients experience chronic pericarditis within 5 to 10 years after radiation therapy.[16] Imaging techniques are frequently utilized in the evaluation of pericarditis to assess the extent of pericardial thickening, the presence and quantification of pericardial effusion, and for monitoring disease progression.[17] Imaging modalities, such as an echocardiogram, cardiac computerized tomography, and cardiac magnetic resonance are useful for establishing a diagnosis and to rule out more serious underlying pathology, for example cardiac tamponade.[16, 17]

Radiation exposure is widely correlated with coronary artery disease (CAD). While the exact mechanism is still under investigation, coronary vascular damage is thought to be a consequence of an increased production of free radicals, an increase in vascular permeability, and the release of inflammatory mediators.[18] Subsequently, intimal proliferation and fibrosis leads to vessel stenosis, as well as the development of clinically significant cardiac complications. The pathological changes associated with radiation-induced coronary artery disease (RICAD) share many histopathologic features with atherosclerosis.[18] In fact, risk factors for developing RICAD remain the same as those associated with non-irradiated CAD.16 Similarly, the diagnostic and management approach to RICAD parallels those with CAD in the general population. Patients with RICAD typically present with angina, dyspnea, and heart failure.[18] Surgical intervention has been shown to be just as effective for RICAD as in atherosclerotic disease.[13] In addition, both coronary artery bypass graft and percutaneous intervention have been widely employed in the treatment of appropriately selected patients.[16] Thus, clinical knowledge regarding this late complication has important implications in reducing the incidence of severe consequences, such as stroke or myocardial infarct since successful treatment modalities are available. From a primary care standpoint, long-term cardiovascular follow-up is essential in reducing negative outcomes in these patients.

DERMATOLOGIC TOXICITY

Radiation dermatitis is one of the most common adverse effects associated with radiation therapy for breast, perineal, and prostate cancers.[3] Despite modern radiation techniques, it is estimated that as many as 90% of patients that undergo radiation therapy develop a skin reaction.[19] Patients with radiation dermatitis usually develop erythema, itching, telangiectasias, alopecia, and ulcerations.[3] Severe skin reactions may be painful and lead to more serious complications, such as infection, necrosis, and permanent scarring.[20] Radiation dermatitis also carries a significant psychological burden.[19] In addition to the emotional impact of cancer diagnosis and treatment, patients suffering from radiation dermatitis experience a reduced quality of life.[19] It is thought that inflammatory mediators associated with damage to the epidermis contribute to the development of radiation dermatitis. Chen et al. showed that IL-1, an inflammatory cytokine, plays a significant role in modulating skin toxicity in a mouse model for radiation dermatitis.[21] Clinically, acute exposure typically produces symptoms within 10-14 days. It is widely accepted to use moisturizers to reduce skin irritation. In addition, topical steroids are commonly used prophylactically to prevent radiation dermatitis; however, evidence is limited regarding the effectiveness of this therapy.[3,19] Further investigation is necessary to determine the value of topical steroids and other pharmacological agents in the treatment of skin toxicity.

CONCLUSION

Vast improvements in radiation techniques and risk management over the past few decades have led to increased cancer survival rates and reduced radiation-induced adverse effects. While the benefits of using radiation therapy in the treatment of cancer have been well established, the development of acute and chronic adverse effects and their implications on long-term morbidity and mortality remain largely unknown. Literature surrounding late radiation toxicity is limited due to the fact that these adverse affects have only recently become prevalent in an aging population receiving curative treatment for cancer. While the primary care physician may not be directly treating the cancer patient undergoing radiation therapy, there is a strong likelihood that the physician will encounter patients treated in this manner. Developing a plan to identify acute and chronic side effects of radiation therapy is important in the management of the entire patient. Collaboration with the specialist will allow for an optimal care plan for the patient and could minimize patient anxiety and reduce unnecessary diagnostic testing.

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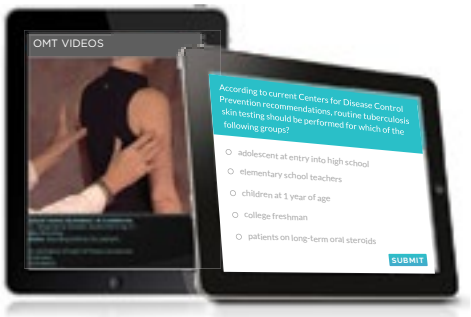

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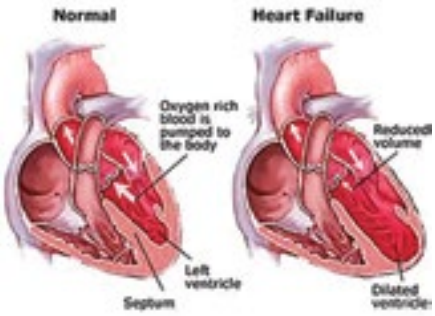

Heart failure is a condition in which the heart does not pump blood as well as it should. It is more often seen in people who are overweight, 65 years of age or older, African Americans, and people who have had a heart attack. Men have a higher rate of heart failure than women do. Common causes of heart failure include high blood pressure, diabetes, prior heart attacks, bad heart valves, damaged heart muscle, and abnormal heart rhythms. Tobacco use, drinking too much alcohol and sleep apnea also increase the risk of heart failure. Signs and symptoms of heart failure may include a buildup of fluid in the lungs, liver, abdomen, feet, ankles, and legs, shortness of breath, feeling tired, chest pain, irregular heartbeat, a persistent cough or wheezing, sudden weight gain from fluid retention, sleep disturbance, increased urination at night, lack of appetite, and nausea.

Preventive Measures Include:

- Maintain a Healthy diet and Weight: Eat a diet that includes fresh fruits and vegetables, whole grains, low fat dairy products and lean proteins. If you are overweight, your doctor can help you work towards a healthy weight.
- Avoid salt in your diet: Too much sodium contributes to water retention, which makes the heart work harder and causes shortness of breath and swollen legs.
- Be active. Your doctor can help develop an exercise plan that is right for you.
- Stop Smoking! Smoking damages blood vessels, increases blood pressure, lowers the amount of oxygen in your blood and makes your heart beat faster. Avoid secondhand smoke also.
- Limit alcohol. If you have heart failure, do not drink alcohol. Alcohol can interact with your medication(s), weaken your heart muscle and increase your risk of abnormal heart rhythms. If you have severe heart failure, your family doctor may also recommend that you limit the amount of fluids you drink.
- Reduce stress and Sleep easy: When you are anxious or upset, your heart beats faster, you breathe more heavily and you blood pressure often rises. Find ways to reduce stress in your life. Spend time with family and friends. Avoid caffeine. If you have sleep problems or snore, make sure you are tested for sleep apnea.
- Consider Vaccinations: If you have heart failure, you may want to get influenza and pneumonia vaccinations.
- If you have a condition such as diabetes or high blood pressure, control your condition by taking your medications regularly as prescribed by your doctor. Before taking any OTC medications or supplements, talk to your doctor.

Medical Care & Treatment Options:

If you have any questions about heart failure, please contact your Osteopathic Family Physician. Your doctor can diagnose heart failure by doing a physical exam along with heart, lung, and blood tests. Management includes the right treatment plan and regular visits with your doctor. Your family doctor will help you choose which drugs and treatments will work best for you. In case of any emergency, you should call your doctor or 911 right away.

Source(s): American Heart Association, Medscape, National Heart, Lung, and Blood Institute, and Prescriber's Letter.

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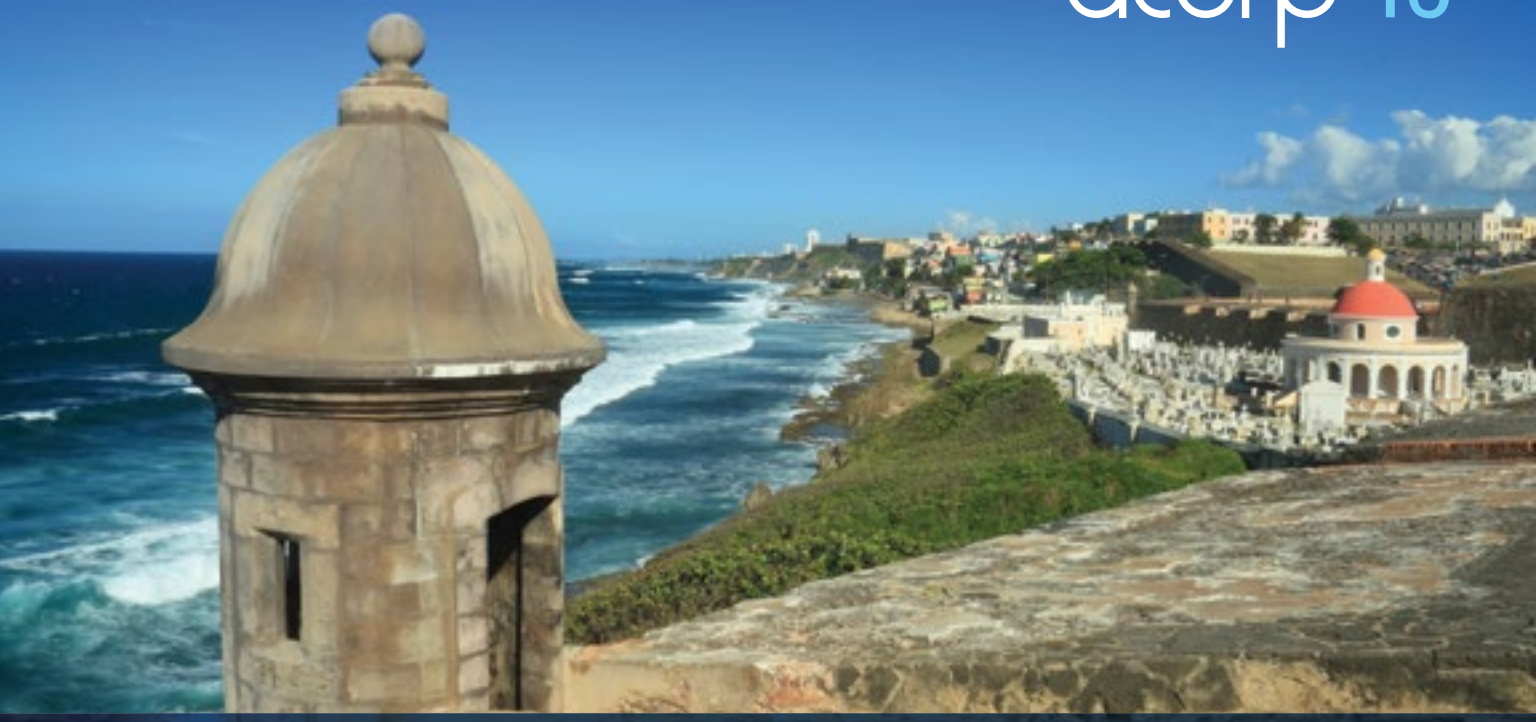

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