Flash Pulmonary Edema in Patients with Chronic Kidney Disease and End Stage Renal Disease

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Flash pulmonary edema and acute pulmonary edema are terms used to define the sudden development of respiratory distress related to the rapid accumulation of fluid within the lung interstitium secondary to elevated cardiac filling pressures (Little, & Braunwald, 1997). For the purposes of this review, flash pulmonary edema will be the term used. One of the first studies to describe its occurrence linked its development to individuals with pre-existing coronary artery disease and hypertension (Lee, Cabin, & Francis, 1988). The connection between flash pulmonary edema and kidney disease was initially described in individuals with bilateral renal artery stenosis (Pickering et al., 1988). This association has been so well characterized that the recommendation has been made that anyone presenting with flash pulmonary edema be considered for evaluation for renal artery stenosis (Missouris, Belli, & MacGregor, 2000).

The risk for flash pulmonary edema in individuals with chronic kidney disease (CKD), primarily end stage renal disease (ESRD), has been under emphasized in the literature. There are several possible explanations for the lack of reports describing an association between flash pulmonary edema and CKD, especially in patients with CKD receiving maintenance dialysis. The sudden onset of pulmonary edema may be assumed to be from excessive interdialytic weight gain, inaccurate dry weight prescription, or weight scale malfunctions rather than from a cardiogenic origin. Furthermore, treatment is often readily available and implemented (ultrafiltration), thus the problem is promptly treated.

The strong association between cardiovascular disease and kidney disease has been emphasized during the past decade (Foley, Parfrey, & Samak, 1998). Kidney disease is now known to be an independent risk factor for cardiovascular morbidity and mortality (Levey et al., 1998; Sarnak et al., 2003). This article will define flash pulmonary edema and factors for its development, emphasizing the relationship between cardiovascular disease and chronic kidney disease. Lastly, a case study of a patient on hemodialysis that developed flash pulmonary edema will be presented.

Goal
Recognize the risk for development of flash pulmonary edema in patients with chronic kidney disease and ESRD.

Objectives
1. Identify causes of flash pulmonary edema that may occur in conjunction with chronic kidney disease and ESRD
2. Recognize signs and symptoms of flash pulmonary edema.
3. Describe nursing measures that may avert development of flash pulmonary edema in individuals with advanced chronic kidney disease.

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The Nephrology Nursing Certification Commission (NNCC) requires 60 contact hours for each recertification period for all nephrology nurses. Forty-five of these 60 hours must be specific to nephrology nursing practice. This CNE article may be applied to the 45 required contact hours in nephrology nursing.
Pathogenesis of Pulmonary Edema

Edema has been described as increased volume within several spaces of the body including the blood vessels (increase in blood volume), the lungs (pulmonary interstitium and alveoli), the trunk and lower extremities (peripheral edema), as well as cavities within the abdomen and lungs (i.e., pleural effusions and ascites). Peripheral edema is most evident in the lower extremities because of the effect of gravity. The occurrence of peripheral edema in patients with CKD may be attributed to either heavy proteinuria (over 3.5 grams per day) or advanced deterioration in kidney function (Bickley, Hoekelman, & Bates, 1999). Pulmonary edema results from fluid accumulation in the lungs at a higher rate than can be removed. The type of abnormality expressed in the Starling equation (Figure 1) differentiates the type of pulmonary edema. It can predict the net flow of fluid across a membrane based upon permeability, surface area, and hydraulic pressures (Hanley & Welsh, 2004).

Pulmonary edema has been classified into two categories dependent upon the underlying cause: cardiogenic and non-cardiogenic (Hanley & Welsh, 2004). Starling forces identified in the equation affect the fluid balance between the interstitium and the vascular bed within the lungs (capillary permeability, capillary surface area, capillary and interstitial fluid hydraulic pressures, capillary and interstitial fluid oncotic pressures, and the pressure differential across the capillary wall). Normally, the alveolar bed within the lung serves to protect the lung from fluid accumulation. Alveoli have very low permeability for fluid and protein. Any fluid filtered into the alveoli is continuously absorbed back into the interstitium by the alveolar epithelial cells and drained away from the pulmonary interstitium by lymphatic vessels (Gluecker et al., 1999).

Non-cardiogenic pulmonary edema occurs as a result of fluid accumulation in the alveoli resulting from a disruption in Starling’s forces secondary to increased capillary permeability. The most common cause of non-cardiogenic pulmonary edema is acute lung injury (ALI) or acute respiratory distress syndrome (ARDS) associated with increased pulmonary capillary permeability. Most often, the concentration of protein within the pulmonary interstitium exceeds 60% of the plasma protein value as compared to less than 45% in cardiogenic pulmonary edema (Hanley & Welsh, 2004). Non-cardiogenic pulmonary edema is diagnosed when there is radiographic evidence of alveolar fluid accumulation without an elevation in the pulmonary wedge pressure greater than 18 mmHg or evidence of congestive heart failure (CHF) on physical examination (Mason, Broaddus, Murray, & Nadel, 2005).

Cardiogenic pulmonary edema is the most common type of pulmonary edema and results from increased left atrial filling pressure (Hanley & Welsh, 2004). In this type of pulmonary edema, the rate of fluid accumulation within the lungs is mainly dependent upon the functional capacity of the lymphatic system of the lungs to remove fluid from the interstitium and alveoli (Mason et al., 2005). As left atrial pressure rises, pulmonary capillary pressures increase resulting in protein poor fluid (edema) being forced into the lung interstitium and alveoli. Consequently, there is a progressive deterioration in alveolar gas exchange resulting in hypoxemia and symptoms of respiratory distress. Compensatory changes may occur in individuals with chronically elevated left atrial pressures and persistently elevated pulmonary capillary wedge pressures (greater than 18 mmHg). In this circumstance, the lymphatic system of the lungs becomes more efficient at removing fluid, thus reducing the likelihood for development of edema. It is the sudden or acute elevations in left atrial pressures that are more likely to result in acute pulmonary edema (Fraser, Muller, Colman, & Peré, 1999; Little, & Braunwald, 1997).

It may be difficult to differentiate between flash pulmonary edema and non-cardiogenic pulmonary edema since the presenting symptoms are often the same (Hanley & Welsh, 2004). Symptoms typically consist of dyspnea, tachypnea, and cough with possible expectoration of frothy edema fluid. The patient history and determination of the factors that led to the development of the pulmonary edema becomes a priority for diagnosis. Non-cardiogenic pulmonary edema is less common and is primarily seen in individuals that have had recent pneumonia, sepsis, tumor, pulmonary fibrosis, trauma with multiple blood transfusions, following lung transplantation or resultant from aspiration of gastric contents.
nervous systems becomes maladaptive by contributing to cardiac myocyte apoptosis (programmed cell death), hypertrophy, and focal myocardial necrosis (Jackson et al., 2000).

Diastolic dysfunction, defined as a disorder of myocardial relaxation resulting in impaired ventricular filling, may also result in the syndrome of congestive heart failure. Diastolic heart failure is diagnosed when elevated filling pressures are necessary to achieve normal ventricular filling (Grossman 2000). Recent epidemiologic data has determined that 40% - 50% of patients presenting with congestive heart failure have diastolic dysfunction as the predominant etiology. The diagnosis of diastolic heart failure is made based upon the existence of heart failure in patients with normal systolic function (left ventricular ejection fraction of 50% or greater) and no evidence of valvular or pericardial disease (Vasan, Benjamin, & Levy 1995). Some individuals will have evidence of both systolic and diastolic heart failure contributing to development of flash pulmonary edema.
edema. However, diastolic heart failure occurs more frequently in patients with preserved systolic function, thus it has been attributed primarily to the disorder of diastolic dysfunction (Gandhi et al., 2001).

Although diastole involves the process of cardiac relaxation, active energy requiring processes occur during this phase. The changes in cardiac pressure that occur during diastole are the result of isovolumetric relaxation from the time of the aortic valve closure to mitral valve opening; early rapid filling after mitral valve opening; and low blood flow during mid-diastole; and lastly late filling [high blood flow] from atrial contraction. In diastolic heart failure, the left ventricle is stiff [reduced elastic recoil] with impaired relaxation causing a reduction in filling. Higher diastolic pressures are required to maintain adequate ventricular filling (Redfield, 2004).

The literature has shown a strong association between hypertension and development of diastolic heart failure, as well as a strong association with development of flash pulmonary edema. One study described the presence of systolic hypertension (systolic arterial blood pressure greater than 160 mmHg) in 85% of patients presenting to hospital emergency rooms with flash pulmonary edema (Kramer, Kirkman, Kitzman, & Little, 2000). Studies that performed Doppler echocardiography after the acute episode found preserved systolic function [left ventricular ejection fraction of 50% or greater] in the majority of patients presenting with flash pulmonary edema. Even though systolic function was assessed as normal, the evaluation was obtained after treatment of the hypertensive episode and resolution of the pulmonary edema, thus the presence of transient systolic dysfunction as a precipitating cause could not be excluded (Mansoor et al., 2001). The primary cause for flash pulmonary edema, systolic versus diastolic heart failure, was addressed in a study that examined patients with two-dimensional transthoracic echocardiography using color Doppler imaging during the acute phase of pulmonary edema with a follow up exam performed 2-3 days later. The left ventricular ejection fraction during the acute episode was similar to the measurement obtained 2-3 days after presentation (N = 30). Thus, a normal left ventricular ejection fraction in a patient with co-existing hypertension and flash pulmonary edema suggests that the pulmonary edema is due to diastolic dysfunction. Transient systolic dysfunction and severe mitral regurgitation were found to be infrequent causes for development of flash pulmonary edema (Gandhi et al., 2001).

Other clinical conditions that have been identified as contributing to the development of flash pulmonary edema include: a) myocardial ischemia, b) acute aortic insufficiency, c) acute mitral regurgitation, d) mitral stenosis, and e) renovascular hypertension (Walker, Walker, & Nielsen, 2001), but this paper will primarily focus on diastolic heart failure as it relates to development of flash pulmonary edema because diastolic heart failure has received less attention and yet is believed to be highly prevalent.

Cardiac Disease

Flash pulmonary edema occurs as a consequence of a disruption in the normal pressure-volume relationship during the cardiac cycle. Ischemic heart disease/coronary artery disease has been linked with development of flash pulmonary edema. Acute myocardial ischemia has been shown to cause systolic and diastolic dysfunction. Cocaine abuse has been associated with development of flash pulmonary edema for multiple reasons including its precipitation of myocardial ischemia [coronary artery vasoconstriction], acute rise in blood pressure [peripheral vasoconstriction], and development of systolic as well as diastolic dysfunction (Lange & Hillis, 2001).

The existence of coronary artery disease can cause intermittent episodes of diastolic dysfunction and systolic dysfunction (Mansoor et al., 2001). Systolic dysfunction occurs when there is less forward movement of blood from the heart prompting an increase in diastolic volume and diastolic pressure precipitating pulmonary vascular congestion leading to pulmonary edema. In diastolic heart failure, the myocardium is less compliant, such that the left ventricle is unable to accept an adequate volume of blood from the venous system and to fill at normal low pressures (Beattie, 2000). The net result is a rise in diastolic pressures in order to provide adequate ventricular filling. Heart failure resulting from diastolic dysfunction occurs when elevated filling pressures are necessary to achieve normal ventricular filling (Grossman, 2000). The increased resistance to diastolic ventricular filling is most commonly due to myocardial abnormalities (myocardial hypertrophy, fibrosis, ischemia, or cardiomyopathy), and less commonly to mechanical abnormalities like mitral stenosis or constrictive pericarditis (Hanley & Welsh, 2004).

Hypertension is frequently present in patients presenting with flash pulmonary edema. Chronic hypertension that is poorly controlled can result in development of systolic and diastolic dysfunction that may predispose patients to the development of flash pulmonary edema (Mansoor et al., 2001). Even labile elevations in blood pressures of patients with bilateral renal artery stenosis have been shown to be associated with an increased risk for development of flash pulmonary edema (Bloch, Trost, Pickering, & Sos, 1999). As noted by Vasan & Levy (1996) long standing hypertension is a primary predis-
logic hypertrophy of the left ventricle and maintain near or near-normal exercise tolerance. Therefore, LVH by itself does not constitute cardiac dysfunction (Lorell & Carabello, 2000).

**Renal Artery Stenosis and Flash Pulmonary Edema**

Renal artery stenosis (RAS) may be identified in individuals with apparent normal kidney function as well as those with a diagnosis of acute or chronic kidney disease (Missouris, Belli, & MacGregor, 2000). Renal artery stenosis (narrowing of the renal artery) is most often a result of atherosclerosis. Fibromuscular dysplasia, is a rare idiopathic condition in which there is an increase in the number of cells in the renal artery cell wall resulting in arterial narrowing. Atherosclerotic lesions result from the deposition of plaque within the renal artery and are most often seen in individuals over the age of 55, while fibromuscular dysplasia is usually seen in young women. Women with fibromuscular dysplasia associated RAS almost invariably have hypertension. Lesions associated with fibromuscular dysplasia tend to show a much more favorable response to percutaneous transluminal angioplasty (PTA), stent or surgery than atherosclerotic lesions (Olin, 2004).

The most common type of RAS involves atherosclerotic deposition in the artery lumen that disrupts blood flow to the kidney parenchyma causing ischemia. The kidney responds by secreting renin into the bloodstream, leading to the production of angiotensin II, which is both a potent vasoconstrictor and a major stimulus for adrenal secretion of aldosterone (Safian & Textor, 2001). Hypertension resulting from activation of the RAS is referred to as renovascular hypertension. Mild to moderate hypertension is not commonly associated with RAS; however, malignant and drug resistant hypertension carries a high degree of suspicion for RAS, having been diagnosed in up to a third of patients having difficult to control or malignant hypertension (Harding et al., 1992). Renovascular hypertension related to bilateral renal artery stenosis is strongly associated with the development of flash pulmonary edema. The combination of hypertension and volume retention, occurring as a consequence of altered intrarenal hemodynamics and aldosterone-mediated salt and water retention are the major contributing factors to the development of flash pulmonary edema with bilateral renal artery stenosis. In contrast pulmonary edema is relatively uncommon with unilateral renal artery stenosis (Basaria & Fred, 2002; Jaff, 2001). Unilateral renal artery stenosis leads to renin dependent hypertension, but not to volume expansion, since the non-affected kidney undergoes a pressure natriuresis/diuresis, which prevents volume expansion making pulmonary edema less likely to occur (Missouris et al., 2000).

**Flash Pulmonary Edema and Kidney Disease**

Many of the precipitating factors associated with development of flash pulmonary edema are frequently present in patients with advanced stages of CKD (Table 1). Cardiovascular disease and CKD are strongly linked. Recent findings indicate that patients with CKD are in the highest risk group for cardiovascular disease and that patients with CKD are more likely to die of cardiovascular disease than ESRD (Levin, 2003).

Systolic and diastolic heart failure has been associated with the development of flash pulmonary edema (Ware & Matthay, 2005). This discussion will primarily highlight the pathology of diastolic heart failure. A relationship between the risk for diastolic heart failure in the presence of kidney disease can be established by examining the contributing factors that precede there development (Figure 3). Diastolic heart failure can be caused by ischemia, left ventricular hypertrophy (LVH), increased filling pressures related to chronically elevated blood pressures, infiltrative cardiomyopathies, ischemic heart disease, pericarditis, the normal aging processes, chemotherapy and genetic anomalies (Beattie, 2000; Kramer et al., 2000; Zile & Simsic, 2000). In the general population, 50% of people over the age of 70 with a diagnosis of congestive heart failure will have preserved systolic function and classified as having diastolic heart failure. For those 10 years younger (60 years of age) with congestive heart failure symptoms, only 15% will have diastolic heart failure. Age also contributes to mortality risk. Overall, prognosis is better for diastolic heart failure (average mortality rate 5 % - 8%) than for systolic heart failure (average mortality rate 10% - 15%), but diastolic heart failure in patients over 70 years has a 50% 5-year mortality rate (Zile & Simsic, 2000). These changes are similar to disease reported in the elderly, but they occur 15-20 years earlier in kidney disease (Tozawa, Iseki, Iseki, & Takishita, 2002; Churchill et al., 1992). Systolic

Table 1: Prevalence of Risk Factors: Diastolic Heart Failure in CKD

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<thead>
<tr>
<th>Risk</th>
<th>Prevalence</th>
<th>References</th>
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<tr>
<td>Hypertension</td>
<td>50% - 75%</td>
<td>NKF, 2005</td>
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<tr>
<td>LVH</td>
<td>75% - Beginning dialysis</td>
<td>Levin, 2003</td>
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<tr>
<td>Age</td>
<td>Average age of dialysis</td>
<td>USRDS, 2005</td>
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<tr>
<td>Ischemic heart disease</td>
<td>35% - CKD</td>
<td>Levin, 2003</td>
</tr>
<tr>
<td>Heart failure</td>
<td>40% - Beginning dialysis</td>
<td>Foley et al., 1994</td>
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Pulmonary Edema

Renal Artery Stenosis and Flash Pulmonary Edema

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<tr>
<td>Age</td>
<td>Average age of dialysis patient – 63 years</td>
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<tr>
<td>Ischemic heart disease</td>
<td>35% - CKD</td>
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<tr>
<td>Heart failure</td>
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and diastolic heart failure are highly prevalent in patients with advanced CKD (Foley et al., 1994). Increasing emphasis has been placed on the need to control blood pressures in all stages of CKD. Since hypertension is both a cause and a complication of CKD, prevalence rates are high. Elevated blood pressure is a primary contributing factor to development of LVH and heart failure (both systolic and diastolic). Lucas and colleagues (2003) found that systolic hypertension and increased pulse pressures are common at the time of dialysis initiation. All cause mortality was significantly higher, 14.1 deaths per 100 patient-years for patients with uncontrolled hypertension (BP greater than 140/90 mmHg) compared to those that were normotensive, 7.9 deaths per 100 patient years (RR = 1.79, P = 0.01, 95% CI = 1.15–2.8.). Blood pressure control remains poor even after beginning renal replacement therapy. Reports indicate that blood pressure control in patients receiving dialysis is low with 40% – 90% having blood pressures higher than the National Kidney Foundations K-DOQI recommended goal of 140/90 pre-dialysis (Chalmers et al., 1999; Dhakal, Sloand, & Schiff, 2000; Salem, 1995).

Coronary artery disease has also been recognized as a contributing factor to development of flash pulmonary edema. Chronically elevated blood pressure results in stiff arteries that have diffuse arteriosclerosis. Murphy (2003) reports a coronary artery disease prevalence rate of 40% in patients beginning dialysis, 22% have stable angina and 8% have historical documentation of a previous myocardial infarction.

The diagnosis of flash pulmonary edema is made using a compilation of clinical findings (Mansoor, et al., 2001). Initial evaluation should include a chest x-ray and Doppler echocardiogram. Chest radiographs characteristically demonstrate enlargement of the peribronchovascular spaces, prominent septal lines as well as acinar areas of increased opacity that coalesce into frank consolidations.
[Ware & Matthay, 2005]. It is often difficult to differentiate between systolic and diastolic heart failure based upon patient history, physical exam, chest radiograph, and EKG. Doppler echocardiography is the most commonly utilized technique to assess both systolic and diastolic ventricular function. Diastolic function involves both active and passive components. The active phase involves energy dependent relaxation while the passive phase predominantly reflects the viscoelastic properties of the heart tissue.

Doppler echocardiography assesses both of these phases by examining pressure changes during transmitral flow (Figure 4) in diastole. Early diastolic flow occurs when the mitral valve initially opens at the onset of diastole. The E wave determined by Doppler echocardiography serves as an index of left ventricular relaxation, compliance, and atrial pressure. Early passive ventricular filling is followed by the ventricular filling produced by atrial contraction (A wave). Deceleration time (DT) is a measurement of the time it takes for passive filling. Deceleration time will shorten when there is a decrease in left ventricular compliance however deceleration times are also affected by other pathologies that increase the left atrial pressure and shorten DT. DT values over 240 ms indicate impaired relaxation, and under 150 ms indicate a restrictive pattern (Gilbert, Connelly, Kelly, Pollock, & Krum, 2006).

Interpretation of E:A ratios becomes more difficult when there are combined pathologies of impaired relaxation and restriction, then both left atrial and ventricular pressures increase leading to an increase in the magnitude of the E wave resulting in a normal E:A ratio (pseudonormal pattern) which can be unmasked by maneuvers that alter cardiac preload such as the Valsalva maneuver or administration of nitroprusside (Angeja & Grossan, 2003). Even though there are problems with interpretation of E:A ratios, transmitral inflow patterns obtained with Doppler echocardiography are non-invasive and can offer further support in determining the existence of diastolic heart failure, especially when there is isolated maladaptive relaxation of the ventricles.

If the Doppler echocardiogram is non-conclusive or technically inadequate due to a poor acoustic window, then a multiple-gated acquisition (MUGA) scan otherwise known as a cardiac blood pool scan may be required. This scan uses a radioactive isotope to evaluate ventricular function and detect abnormalities in the heart wall (Logeart et al., 2002). The MUGA is more accurate at calculating ejection fraction, but less efficient at identifying valve morphology. The radio isotope (technetium) is injected into a vein and absorbed immediately by healthy tissue. A gamma scintillation camera detects the gamma rays emitted by the radio isotope. If the patient’s heart is normal, the technetium will be readily and evenly distributed in the cardiac images. An uneven distribution of technetium in the heart may be indicative of coronary artery disease, cardiomyopathy, or blood shunting within the heart. Use of the gamma scintillation camera exposes the patient to about the same amount of radiation as a chest x-ray (Youngerman-Cole, 2005).

Even though rarely done, the gold standard for the diagnosis of diastolic function involves the measurement of transmitral pressures as described in the text. Figure 4 illustrates the normal E:A ratio and deceleration time, as well as the restrictive and pseudonormal E:A ratios and deceleration times.
Heart failure continues to be cardiac catheterization allowing direct measurement of volumes and pressures, but at significantly higher cost and risk (Gutierrez & Blanchard, 2004). It is usually not necessary to determine the etiology for the heart failure since Doppler echocardiogram or MUGA usually provide adequate diagnostic criteria. Pulmonary artery catheterization can also be done and has an adverse event rate of 4.5% - 9.5%, but it is much more precise in defining the presence of diastolic heart failure than MUGA or Doppler echocardiogram alone. If the pulmonary artery occlusion pressure is greater than 18 mm Hg then cardiogenic pulmonary edema exists (Harvey et al., 2005).

Laboratory testing in flash pulmonary edema typically may include measurement of plasma levels of troponins and brain natriuretic peptide (BNP). Troponin I elevation in the presence of kidney failure has demonstrated high prognostic value for acute coronary syndrome (myocardial ischemia). Troponin T elevation has not been recommended for detecting acute myocardial ischemia, but has been associated with increased mortality risk (Freda, Tang, Van Lente, Peacock, & Francis, 2002). In a recent study of 258 hemodialysis patients, elevations in Troponin T (0.10 ng/mL or greater) correlated significantly with the presence of left ventricular hypertrophy (Iliou et al., 2001). The rise and fall of Troponin I (trend) may be required to make a definitive diagnosis of acute coronary syndrome since 1% - 6% have been shown to have elevations without symptoms (Freda et al., 2002).

Elevations in brain natriuretic peptide (BNP) have been routinely used in the diagnosis of heart failure. When the ventricular wall stretches or there is increased ventricular pressure, BNP is secreted. A recent consensus panel concluded that BNP levels below 100 pg/mL indicate that heart failure is unlikely (negative predictive value, greater than 90%); while a BNP level greater than 500 pg/ml indicates that heart failure is likely (positive predictive value, greater than 90%) (Silver et al., 2004). Intermediate levels of BNP, between 100 and 500 pg/mL had less diagnostic discrimination. BNP levels may be elevated in critically ill patients, even in the absence of heart failure; however, BNP levels less than 100 pg/ml continue to be useful in excluding heart failure in these patients. BNP levels are also elevated in patients with chronic kidney disease, such that a cut off level below 200 pg/mL has been suggested to exclude heart failure when the estimated glomerular filtration rate is below 60 mL per minute. Chronic elevations in BNP may also be a hallmark of patients with or at risk for diastolic heart failure among subjects with preserved systolic function, independent of the degree of left ventricular hypertrophy (Yamaguchi et al., 2004).

Symptoms of Flash Pulmonary Edema

Complaints of severe cough and dyspnea are usually the primary symptoms on presentation of flash pulmonary edema with or without chest discomfort. Common findings on physical examination include:

- Tachypnea – with use of accessory muscles of breathing.
- Lung fields – auscultation of crackles in bases and scattered throughout the lung fields, rales or even decreased breath sounds.
- Cardiac exam – possible presence of an S3 indicating an increase in left ventricular end diastolic volume or an S4 gallop coinciding with an increase in left atrial pressure, and a new or changed murmur.
- Jugular venous distension indicating increased filling pressures.
- Systolic Blood pressure – may be markedly elevated. Hypertension suggests left ventricular systolic dysfunction and impending cardiogenic shock.
- Peripheral edema – usually without signs of edema, if present, then associated with long-standing volume expansion (Mason, Broaddus, Murray, & Nadel, 2005).

Flash Pulmonary Edema: A Case Presentation

A 54-year-old male presented to the dialysis unit of an acute care hospital due to inadvertent dislodgement of his tunneled venous catheter. He had been receiving chronic hemodialysis for 3 years at the time of presentation and was oliguric. Arrangements were made with the interventional radiology department to have this catheter removed and a new catheter placed. He had undergone hemodialysis at the community dialysis center 48 hours prior to his presentation.

There was no evidence of cardiopulmonary compromise on admission and he was able to ambulate to the unit without difficulty. His blood pressure was noticeably elevated and he reported holding his antihypertensives that day in anticipation of undergoing hemodialysis. Holding his antihypertensives on the day of dialysis was a common practice. A chest radiograph was obtained prior to sending the patient to interventional radiology to evaluate for any evidence of volume expansion since he would most likely miss his dialysis treatment that day (see Figure 5).

Physical examination on presentation:

- Temperature: 97.8°F;
- Weight: 71 Kg (less than 2 kg above present dry weight estimate);
- Blood pressure: 165/116 HR 89 (sitting); blood pressure 151/88 HR 86 (standing);
- No evidence of jugular venous distension;
- Cardiovascular exam: Regular rhythm, rate 89/minute, 1/VI systolic murmur, no pericardial rub, no gallops;
- Lungs: Respirations were regular and unlabored, no tachypnea noted, few scattered crackles in lung bases, otherwise
clear to auscultation; and
• No peripheral edema present.
Past medical history was significant for:
• Coronary artery disease and myocardial infarction – with coronary artery stent placement 8 months prior,
• Long-standing hypertension,
• Hyperlipidemia.
A 2-D Echocardiogram obtained 3 months earlier demonstrated:
• Diffuse hypokinesia of the left ventricle, with an approximate ejection fraction of 29%;
• Left ventricular diastolic dysfunction;
• Mild eccentric left ventricular hypertrophy;
• Trace tricuspid regurgitation;
• No pericardial effusion.
The patient returned from the interventional radiology department following catheter removal and placement of a new cuffed hemodialysis catheter on a stretcher. He was noted to be in acute respiratory distress with the head of the stretcher at 90º.

The following findings were noted upon his return to the dialysis unit.
• Temperature: 97.6º F;
• Blood pressure: 247/142 HR 127 (sitting);
• Pulse oxygenation: 67 – 78% (room air);
• Respiratory rate: 30/minute with use of accessory muscles;
• Productive cough of frothy white sputum;
• Jugular venous distention was present;
• Cardiovascular exam - tachycardia, II/VI systolic murmur, loud S2, positive gallop rhythm, no pericardial rub;
• Lungs - decreased breath sounds in lung bases;
• No peripheral edema noted;
• 12 Lead EKG – Sinus tachycardia with rate 127/minute, occasional premature ventricular complexes, left atrial enlargement, left ventricular hypertrophy and non-specific ST segment changes;
• Portable chest x-ray demonstrated a new dialysis catheter in place in the left internal jugular vein with the tip in the superior vena cava. The previously placed catheter had been removed. There was diffuse bilateral pulmonary interstitial edema (see Figure 6);
• Serial Troponin I: 0.0 – 1.0 ng/mL over 24 hours (Range: 0 – 0.6 – no cardiac damage, 0.7 – 1.5 – non-diagnostic, greater than 1.5 Evidence of a myocardial infarction)
Other lab results included:
• Platelets: 374 K/uL
• Na: 140 meq/dL
• Potassium: 4.4 meq/dL
• HCO3: 39 meq/L
• BUN: 40 mg/dL
• Creatinine: 9.8 mg/dL
• Glucose: 84 mg/dL
• Calcium: 10.8 mg/dL
The treatment included:
• Oxygen by non-rebreather facemask at 60%;
• Nitroglycerin paste: 1” to chest;
• Labetalol: 20 mg IV over 2 minutes; and
• Acute hemodialysis with volume removal.
Hemodialysis was initiated with ultrafiltration of 3.5 liters over a 4-hour period. The patient’s blood pressure improved, and he did not require endotrachial intubation. The oxygen by non-rebreather mask was slowly weaned during dialysis to a nasal cannula oxygen by the end of the dialysis treatment. He was admitted for observation and serial troponins to rule out myocardial infarction, all of which were negative. He returned home after 24 hours with instructions to take his antihypertensive medications the night before dialysis to avoid the surge in blood pressure the morning prior to his hemodialysis treatments.

**Nursing Implications**

Pulmonary edema is a common complication of patients with ESRD (Evans, Reddan, & Szczep, 2004; Shapiro, Deshetler, & Stockard, 1994). Fluid and salt abuse has been reported to be the most common cause of pulmonary edema in patients receiving renal replacement therapy. How does one differentiate between pulmonary edema that is associated with excessive fluid intake and cardiogenic mediated flash (acute) pulmonary edema? Pulmonary edema that occurs without significant interdialytic weight gain or evidence of peripheral edema on physical examination, particularly in association with an elevation in blood pressure, strongly supports the diagnosis of flash pulmonary edema.

A history of ischemic heart disease, poorly controlled hypertension, and identification of left ventricular hypertrophy by Doppler echocardiography may signify an increased risk for development of flash pulmonary edema. Anemia and hypertension have been primarily associated with the development of left ventricular hypertrophy and are found in the majority of patients beginning dialysis. Poorly controlled hypertension in the presence of a remodeled heart (i.e., LVH) may result in diastolic dysfunction which predisposes to the development of pulmonary edema during episodes of volume expansion. The routine practice of holding antihypertensives prior to undergoing hemodialysis in certain circumstances may need to be reconsidered. The practice of holding antihypertensives is to avoid low blood pressures at the end of dialysis following volume removal (Coomer, Schulman, Breyer, & Shyr 1997). Blood pressures tend to normalize with the removal of intravascular volume during the ultrafiltration process. It may be better to reduce rather than hold antihypertensives prior to dialysis to avoid such extremes in blood pressure (Sulkova & Valek, 1988).

The immediate treatment of patients with flash pulmonary edema is essentially the same as with any other type of pulmonary edema. Initial assessment should include monitoring of hemodynamic parameters and pulse oximetry or arterial blood gases. Patients should be given oxygen. This oxygen support should be provided by a non-rebreather face-mask or by positive pressure ventilation. If the work of breathing (respiratory rate greater than 30) and hypoxia is excessive (PaO₂:FiO₂ less than 200) endotracheal intubation may be required (Antonelli et al., 2001). When hypertension is felt to be contributory, obtaining immediate reduction in blood pressure is paramount. Arrangement for acute dialysis with ultrafiltration if the patient has already been receiving dialysis is most beneficial.

Medications commonly used in the treatment of flash pulmonary edema include a) nitroglycerin, b) furosemide, and c) morphine sulfate. Nitroglycerin is a potent vasodilating drug which increases cardiac output while decreasing preload and afterload. Nitroglycerin reduces pulmonary edema mainly through venous dilatation. Patients receiving dialysis who are oliguric or anuric may have a mild venodilatory response to furosemide, but will not have an effective diuresis. The benefit of morphine in treatment of pulmonary edema has remained controversial. Morphine will reduce blood pressure and pulmonary edema through venous dilatation, but morphine may also cause respiratory depression (Beattie, 2000). Lowering the blood pressure is a priority with severely elevated blood pressures. Labetalol is an effective antihypertensive to use in hypertensive crisis since it can be given intravenously without renal adjustment (NHLBI, 2003). It can be given as a slow intravenous push starting at 20 mg over 2 minutes: additional 2 mg dosages can be given every minute up to 300 mg maximum dose to obtain a gradual reduction in blood pressure (NKF, 2005). Lowering of the blood pressure reduces cardiac filling pressures and allows cardiac output to improve, thus reducing fluid accumulation in the lungs. Additionally slowing of the heart rate will improve diastolic filling, thus improving stroke volume and cardiac output.

Treatment of flash pulmonary edema is aimed at resolving the immediate threat of respiratory distress and hypoxemia and eliminating/treating the underlying cause (i.e., hypertensive crisis). Nurses assess patients’ blood pressures pre-dialysis and post-dialysis, thus they are the first to note changes that may place that patient at risk. If blood pressures are assessed as above the recommended pre-dialysis reading of 140/90, then changes in the treatment regimen can be instituted. It may be necessary to reduce the estimated dry weight to achieve a normalized pre-dialysis pressure or additional antihypertensives may be required. When hemodialysis is postponed or canceled, then nurses need to remind patients to resume their antihypertensives in order to avoid blood pressure extremes and risk of flash pulmonary edema.

**Conclusion**

Patients with CKD are at risk for developing flash pulmonary edema. Cardiovascular disease is highly prevalent in the kidney disease population. Diastolic heart failure is often unsuspected without clinical suspi-
cicn since the presentation is similar to that seen with systolic heart failure. The prevalence of LVH at the time patients begin dialysis increases the likelihood of patients developing both systolic and diastolic dysfunction which can lead to development of flash pulmonary edema. The National Kidney Foundation KDOQI guidelines for cardiovascular disease recommends that echocardiograms be performed on all patients at the initiation of dialysis and again after achievement of prescribed dry weight and at 3 year intervals thereafter (NKF, 2005). Nurses should acknowledge and recommend treatment of blood pressures that are assessed to be elevated beyond the current pre-dialysis blood pressure recommendation of 140/90 pre-dialysis and 130/80 in the CKD population (NKF, 2005). Nurses are often the first to assess blood pressures in patients on dialysis and evaluate response to changes in therapy. Patients may institute better compliance and demonstrate improvements in blood pressure regulation with a greater understanding of the inherent risk. With the high preponderance of patients beginning dialysis with cardiovascular disease, it is imperative that clinicians make a more concerted effort in treating the risks for cardiovascular disease.

References
Flash Pulmonary Edema in Patients with Chronic Kidney Disease and End Stage Renal Disease

study. *Nephrology, Dialysis & Transplantation, 16*(7), 1452-1458.


Flash Pulmonary Edema in Patients with Chronic Kidney Disease and End Stage Renal Disease

Carol Headley, DNsC, RN, CNN and Barry M. Wall MD

Posttest – 1.5 Contact Hours

Posttest Questions

(See posttest instructions on the answer form, on page 28.)

1. What statement is true about the fluid balance between the interstitium and the vascular bed within the lungs?
   A. Alveoli have high permeability for protein and fluid.
   B. Alveoli epithelial cells drain the pulmonary interstitium.
   C. The alveolar bed within the lungs serve to protect the lung from fluid accumulation.
   D. The lymphatic vessels continuously absorb fluid from the alveoli.

2. The most common cause of non-cardiogenic pulmonary edema is
   A. renal artery stenosis.
   B. malignant hypertension.
   C. acute lung injury.
   D. aspiration of gastric contents.

3. What changes likely result in acute cardiogenic pulmonary edema?
   A. The lymphatic system becomes less efficient in removing fluid and there is increased likelihood for development of edema.
   B. Acute elevations in left atrial pressure result in pulmonary capillary pressure increases and fluid is forced into the interstitium and alveoli.
   C. Elevated pulmonary capillary wedge pressure overwhelms the lymphatic system and edema results.
   D. Increased pressure and systolic hypertension leads to decrease cardiac output and fluid leaks into the alveoli.

4. Congestive heart failure (CHF) is a disorder that affects the
   A. heart only.
   B. heart and kidneys only.
   C. heart, kidneys, and skeletal muscle only.
   D. heart, kidneys, skeletal muscle and nervous system.

5. What statement is true about neurohormonal changes in congestive heart failure (CHF)?
   A. Initial neurohormonal changes are deleterious and lead to pulmonary edema.
   B. Initially stimulation of the sympathetic nervous system decreases cardiac output.
   C. Stimulation of the RAAS causes salt and water excretion by the kidneys.
   D. Long-term stimulation leads to myocyte apoptosis, hypertrophy, and myocardial necrosis.

6. Diastolic dysfunction refers to a disorder
   A. of myocardial relaxation resulting in impaired ventricular filling.
   B. of myocardial stiffness that results in delayed ventricular emptying.
   C. associated with left ventricular hypertrophy and abnormal filling.
   D. that occurs as a result of pressure changes related to malfunctioning valves.

7. There is a strong association between ———— and the development of diastolic dysfunction and flash pulmonary edema.
   A. hypertension
   B. left ventricular hypertrophy
   C. anemia
   D. volume overload

8. The increased resistance to diastolic ventricular filling in diastolic heart disease is commonly due to
   A. myocardial hypertrophy only.
   B. myocardial hypertrophy and ischemia only.
   C. myocardial hypertrophy, ischemia, and mitral stenosis only.
   D. myocardial hypertrophy, ischemia, mitral stenosis and pericarditis.

9. Which statement is true about the prevalence of diastolic heart failure?
   A. 50% of persons greater than age 60 with CHF will have diastolic failure.
   B. Prognosis for systolic heart failure is better than diastolic heart failure.
   C. Patients over 70 years old with diastolic failure have a 30% 5-year mortality.
   D. Kidney disease contributes to the development of diastolic heart failure at earlier ages.

10. What statement is true about renal artery stenosis (RAS)?
    A. Chronic mild to moderate hypertension is associated with RAS.
    B. It is seen in older women with fibromuscular dysplasia.
    C. It commonly involves atherosclerotic plaque deposition in the renal artery.
    D. It is always associated with a decrease in kidney function.

11. Your patient has a brain natriuretic peptide (BNP) level 700 pg/ml. What would your next action be?
    A. CT scan of the brain
    B. X-ray of the chest
    C. Administer hypertensive medications
    D. Complete a physical assessment

12. Common finding(s) on physical examination of a patient with flash pulmonary edema is (are)
    A. tachypnea only.
    B. tachypnea and decreased breath sounds only.
    C. tachypnea, decreased breath sounds, and new murmur only.
    D. tachypnea, decreased breath sounds, new murmur, and hypotension.

13. What common practice in dialysis units may contribute to flash pulmonary edema?
    A. Holding BP medications pre-dialysis
    B. Inaccurate dry weight assessment
    C. Use of non-tunneled catheters for dialysis
    D. Eating on dialysis

14. Your patient arrives for dialysis with tachypnea, hypertension, and crackles in the bases of the lungs. You feel the patient has cardiogenic mediated flash pulmonary edema. Why?
    A. Insignificant intradialytic weight gain only
    B. Insignificant intradialytic weight gain and absence of edema only
    C. Insignificant intradialytic weight gain, absence of edema, and hypertension only
    D. Insignificant intradialytic weight gain, absence of edema, and hypertension and no history of ischemic heart disease.
ANSWER/EVALUATION FORM

Flash Pulmonary Edema in Patients with Chronic Kidney Disease and End Stage Renal Disease

Carol M. Headley, DNSc, RN, and Barry M. Wall MD

POSTTEST INSTRUCTIONS

- Select the best answer and circle the appropriate letter on the answer grid below.
- Complete the evaluation.
- Send only the answer form to the ANNA National Office; East Holly Avenue Box 56; Pitman, NJ 08071-0056; or fax this form to (856) 589-7463.
- Enclose a check or money order payable to ANNA. Fees listed in payment section.
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CNN: ___ Yes ___ No CDN: ___ Yes ___ No CCHT: ___ Yes ___ No

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1. a b c d 4. a b c d 7. a b c d 10. a b c d 13. a b c d
2. a b c d 5. a b c d 8. a b c d 11. a b c d 14. a b c d
3. a b c d 6. a b c d 9. a b c d 12. a b c d

Evaluation | Strongly disagree | Strongly agree
--- | --- | ---
1. The objectives were related to the goal. | 1 | 2 | 3 | 4 | 5
2. Objectives were met | 1 | 2 | 3 | 4 | 5
   a. Identify causes of flash pulmonary edema that may occur in conjunction with chronic kidney disease and ESRD. | | | | | |
   b. Recognize signs and symptoms of flash pulmonary edema. | 1 | 2 | 3 | 4 | 5
   c. Describe nursing measures that may avert development of flash pulmonary edema in individuals with advanced chronic kidney disease. | 1 | 2 | 3 | 4 | 5
3. The content was current and relevant. | 1 | 2 | 3 | 4 | 5
4. This was an effective method to learn this content. | 1 | 2 | 3 | 4 | 5
5. Time required to complete reading assignment: _________ minutes.

I verify that I have completed this activity:

__________________________
(Signature)

Comments______________________________________________________________
Suggested topics for future articles?______________________________________