Case Study

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History and Presenting Symptoms

An eighty-six year old African American male was admitted to the emergency room on September 26, 2010 for changes in level of consciousness. He had been living in a skilled nursing facility since July. Before living at the skilled nursing facility, he had been living at home with his spouse when he had an acute myocardial infarction, resulting in an anoxic brain injury. There was some debate whether there had really been a change in level of consciousness because he was ventilator dependant, was nonverbal, and only moved his body when he withdrew from pain. Additionally, this patient had a history of atrial fibrillation, gastroesophageal reflux disease, type 2 diabetes mellitus, MRSA of the blood in which he was on contact isolation, renal insufficiency, epilepsy, and multiple decubitis ulcers.

When he was presented to the emergency room, records show that he was non-verbal, ventilator dependant with a trachestomy, withdrew from painful stimulus, and he had spontaneous eye opening. He was suffering from oliguria and emitted dark, cloudy urine with sediment. He was afebrile at 98.2 and had multiple decubitis ulcers in various stages. A PEG tube was present and patent. Arterial blood gases measured at a pH 7.27, O2 98%, HCO3 19.1, PCO2 36.2, showed that the patient was suffering from metabolic acidosis and the BUN 110 and creatinine 3.9 were elevated, revealing renal failure, and there was some thought that he had a gastrointestinal bleed due to a low hematocrit and hemoglobin at 8.7 and 26.6. He was transferred to the intensive care unit, bed four.

The patient’s history stated that he had a pacemaker, but it could not be palpated through the skin and there was no pacer spike in the electrocardiogram. Additionally, there was no evidence of a pacemaker on the chest x-ray, thus leading the doctors to believe that it had been removed. There was a scar on the chest.
There were also scars on the bilateral knees but there was nothing in the history about knee surgery. The patient was nonverbal and there was a language barrier with the wife who was Chinese and could not recall if he had surgery to the knees.

Physical Assessment:

Patient was lying in bed with head of bed increased to 30 degrees with a left wedge tilt. Side rails x4 for safety.

Neurological: [opens] eyes spontaneously, but does not track movement. Withdraws from painful stimuli in upper extremities but unable to move lower extremities. The patient was nonverbal. I was unable to assess bilateral grip strength or strength in legs. Pupils were equal round and reactive to light. Keppra 1000mg PEG bid ordered for history of epilepsy. Ativan 2mg IV/PO q10h PRN for agitation. Chronic renal failure leads to encephalopathy, peripheral neuropathy, loss of motor coordination, twitching, fasciculation of muscles, stupor, and coma due to a progressive accumulation of uremic toxins associated with end-stage renal disease.

Cardiac: S1 and S2 auscultated via stethoscope and noted to be regular with no murmurs. There was no jugular venous distention. On telemetry, the patient was running at normal sinus rhythm with a bundle branch block at 90 beats per minute. The radial pulses were palpable at a 2+, the pedal pulses were found by Doppler only. Generalized edema noted. In the upper extremities, edema was noted at a 2+ non-pitting and lower extremities edema was noted at a 3+ with pitting. Ultrasound of the extremities was performed and there was no evidence of deep vein thrombosis. A 2D echo was done but had not been read by the cardiologist. A CBC revealed WBC of 14.29, Hgb 8.7, Hct, 26.6. Heparin 5000 units SQ Q12h was ordered for DVT prophylaxis but held until results from EGD. For hypertension, Coreg 3.125mg PEG bid,
Lisinopril 5mg PEG qday were ordered. Amioderone 200mg PEG bid for heart rhythm was ordered. Chronic renal failure can lead to left ventricular hypertrophy, hypertension, cardiomyopathy, ischemic heart disease; arrhythmias and pericardial friction rub due to the extracellular volume expansion and hyperexcretion of rennin. Anemia increases cardiac workload.

**Respiratory:** The patient had a chronic tracheostomy with a #8 Schiley trach and after he was suctioned, a small amount of brown, thin secretions were visualized. However, the patient tolerated this procedure well. The ventilator was an assist control and settings are as follows: TV: 550, FiO2 30, Rate 12, PEEP of 3. Rhoncii heard in upper lung fields and diminished heard in bases. A chest X-ray showed pulmonary edema and/or pneumonia. Chronic renal failure can lead to pulmonary edema due to the potential fluid overload of the kidneys not eliminating properly.

**Gastrointestinal:** bowel sounds auscultated in all four quadrants noted to be hypoactive. Abdomen distended non-tender, and soft. Incontinence of bowels during the night shift per report with a positive occult blood in stool. PEG tube in right upper quadrant dressed with gauze, site without redness and drainage. PEG to low intermittent suction with 50 milliliters of coffee brown drainage visualized. There is a plan for an EGD and evaluation of PEG tube so the patient was NPO, and feedings were being held. Pepcid 20mg IV bid given for history of GERD. For constipation MOM 30ml qday PRN, Ducolax 10mg PR qday PRN, Fleets enema 1 bottle PR qday PRN. Chronic renal failure leads to anorexia, nausea, vomiting, mouth ulcers, stomatitis, urinous breath, peptic ulcers, gastrointestinal bleeding, and pancreatitis due to retention of metabolic acids and other metabolic wastes products.
Renal, GU: Foley catheter was in place and yielded 40 milliliters of dark, cloudy urine with sediment. A urine specimen was obtained using aseptic technique. Testicular swelling visualized, otherwise anatomy appeared unremarkable. Lab values were as follows: BUN 110, creatinine 3.9, potassium 4.5, chloride 98. CO2 18, sodium 128 which shows renal failure, ABG pH 7.35, HCO3 19.1, PCO2 34.9, PO2 110.2, O2 saturation 98% on current ventilator settings showing a resolving metabolic acidosis. A renal ultrasound was performed showing a 8mm non-obstructive right renal calculus and mild bladder wall thickening. Possible cystitis or bladder wall hypertrophy, cannot exclude malignancy. 3 amps of sodium bicarbonate IV at 100ml/hr for metabolic acidosis an IV lasix infusion of 20mg of lasix per hour was ordered to reduce the edema and fluid overload.

Musculoskeletal: Contractures in all extremities visualized, weakness in all extremities with some resistance noted. Chronic renal failure can cause spontaneous fractures, bone pain, and deformities of the long bone. This problem is due to the effects of osteitis fibrosa and osteomalacia. A small, persistent decrease in the rate of protein synthesis, or increase in protein degradation, could result in a substantial loss of muscle mass and development of more severe chronic bone disease, seen in patients with CRF and end-stage kidney disease. (Koppel et al. 2005)

Integumentry: Skin is warm and dry to touch and color normal for ethnicity. Poor turgor visualized. Multiple decubitis ulcers as follows: Stage I on coccyx dressed with a hydrocolloid dressing. Left heel stage 4 ulcer with a wet to dry dressing, no drainage noted. Right heel ulcer unstageable due to eschar open to air. Right great toe unstageable due to eschar open to air, and left thumb with eschar open to air. Liquid protein 30ml PEG tid, Arginaid 1 packet PEG q day
to aid in wound healing given.

Chronic renal failure can lead to abnormal pigmentation and pruritus due to retention of urochromes.

**Psychosocial:** Not assessed due to patient’s neurological status. Some information was gathered from the spouse, but there was a significant language barrier, and a Chinese interpreter was not available.

**Endocrine:** Patient per history with diabetes mellitus and capillary blood sugars were being done every six hours. The CBS at 0600 was 113, which required no insulin per physician order. Lantus 20 units SQ qHS and a regular insulin sliding scale ordered for blood sugar coverage.

Metabolic acidosis leads to insulin resistance. As CRF progresses, the ability of the kidney to degrade insulin is reduced and the half-life of insulin is prolonged. Thus, individuals with diabetes mellitus need to carefully manage their insulin doses. ([Huether, McCance, 2008](#))

IV access with a central line was put in the right femoral artery with 3 amps of sodium bicarbonate in 0.45% normal saline infusing at 100ml and hour with no difficulty. Dressing clean, dry, intact with a non occlusive dressing. Site without redness, swelling, or bruising. A peripheral IV was in the right hand with a 20 gauge that was saline locked. Site also dressed with a non occlusive dressing that was clean, dry, and intact. Site was also without redness, swelling, or bruising, flushing with ease.

Because the patient’s blood cultures tested positive for MRSA, Levaquin 750mg IVPB qday and Azactam 1 gram IVPB q8h were ordered. This patient also received supplements due to the PEG feeding and increased nutritional support. These supplements are as follows: multivitamin 15ml qday, zinc 220mg qday, vitamin D 2000 units qday, ferrous sulfate 355mg qday, vitamin C 500mg qday, magnesium oxide 400mg bid, oscal 500mg bid.
Pathophysiology:

The initial presentation for many conditions including sepsis, trauma, renal or endocrine emergencies is often dominated by a metabolic acidosis despite these insults having different etiologies and prognoses. It is essential that an appropriate diagnosis of the pathophysiology of acidosis is made, for example identification of sepsis syndrome or liver failure, but this need not always be the same as identification of the individual acid responsible. There is an increasing recognition of the complex interaction that exists between the respiratory, renal and hepatic systems in controlling acid concentrations, and their interaction in disease. (Morris, Low, 2008)

In acidosis, the acid base balance of the blood and other extracellular fluid is upset by an excess of hydrogen ions. Acidosis is not a disease; it is a condition cause by a disorder or pathological process, in this case renal failure. Regardless of its origin, acidosis causes major changes in body function. The main problems are related to the fact that hydrogen ions are positively charged ions. An increase in hydrogen ions creates imbalances of other positively charged electrolytes, especially potassium. These electrolytes imbalances them disrupt the functions of nerves, cardiac muscle, and skeletal muscle. Even slight increases in blood hydrogen ion levels reduce the activity of many hormones and enzymes, leading to death. (Ignatavicius, Workman, 2010)

Chronic renal failure is the progressive loss of renal function over a period of time of months or years; CRF develops as a complication of systemic diseases, such as hypertension or diabetes mellitus which this patient had. CRF decreases the glomerular filtration rate and tubular functions with changes manifest throughout all organ systems. (Huether, McCance, 2008)
Chronic renal failure (CRF) often results in metabolic acidosis, a process that promotes an increase in hydrogen ions in the body. Metabolic acidosis in CRF is the result of a decreased ability to excrete nonvolatile acid and the reduced renal synthesis of bicarbonate. (Koppel et al. 2005)

Nursing Diagnosis and Interventions:

The patient’s adventitious breath sounds, generalized edema, decreased hematocrit and hemoglobin, and oliguria indicated that he was retaining excess fluid related to a compromised renal output. Excess fluid volume related to compromised renal output as evidenced by adventitious breath sounds, generalized edema, decreased hematocrit and hemoglobin, and oliguria.

**Goal:** Client will maintain urine output within 500ml of intake with normal urine osmolality and specific gravity by the end of 9/27/2010.

Interventions:

1. Monitor location and extent of edema; use a millimeter tape in the same area at the same time each day to measure edema in extremities. *Heart failure and renal failure are associated with dependant edema because of increased hydrostatic pressure.*

2. Administer prescribed diuretics as appropriate; check blood pressure before administration before administration to ensure it is adequate. If administering a diuretic IV, note and record urine output after the dose. *Measuring urine output during diuretic therapy is an acceptable way to measure effectiveness of treatment.*
3. Maintain the rate of all IV infusions carefully with an IV pump. *This is done to prevent inadvertent exacerbation of excess fluid volume.*

4. Monitor for side effects of diuretic therapy such as hypovolemia and electrolyte imbalances. *Observe for hyperkalemia in clients receiving a potassium sparing diuretic, especially with the concurrent administration of an ACE inhibitor.* (Ackley, Ludwig, 2008)

Ineffective protection related to abnormal blood profiles (anemia, elevated WBC) related to deficient immunity, immobility, impaired healing.

**Goal:** Client will remain free from any evidence of new bleeding on 9/27/2010.

Interventions:

1. Watch for hematuria, melena, hematemesis, hemoptysis, epistaxis, bleeding from mucosa, petechia, and ecchymoses. *This type of bleeding can be detected in a bleeding disorder.*

2. Take vital signs often; watch for changes associated with fluid volume loss. *Excessive bleeding causes decreased blood pressure and increased pulse and respiratory rates.*

3. To decrease the risk of bleeding, avoid administering salicylates, NSAIDS, or thrombolytics if possible. *These medications can cause GIB, or increase it if suspected.*

4. Monitor the client’s risk for bleeding; evaluate results of clotting studies and platelet counts. *Laboratory studies give a good indication of the seriousness of bleeding potential.* (Ackley, Ludwig, 2008)

Impaired skin integrity related to impaired metabolic state as evidenced by multiple decubitis, dx of metabolic acidosis, and immobility.
**Goal:** Client will begin to regain integrity of the skin surface by discharge from hospital.

**Intervention:**

1. Assess site of skin impairment and determine cause. *The cause of the wound must be determined before appropriate intervention can be implemented.*

2. Monitor site of skin impairment at least once a day for color changes, redness, swelling, warmth, pain, or other signs of infection. *Systematic inspection can identify impending problems early.*

3. Do not position client on the site of skin impairment. If consistent with overall client management goals, reposition client every 2 hours. Transfer the client with care to protect against the adverse effects of external forces such as pressure, friction, and shear. *This will prevent any new skin impairments.*


**Compromised family coping related to inadequate understanding of information by a primary person (spouse) as evidenced by statements made by significant other about her inadequate knowledge base, complaints about patient’s response to health problems, language barrier.**

**Goal:** Spouse will verbalize knowledge and understanding of disease by the end of 9/27/2010

**Interventions:**

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1. Establish rapport with family by providing accurate communication. *Family care can be improved by focusing on building rapport and communicating problems and concerns between health professionals.*

2. Encourage family to verbalize feelings. Spend time with them, sit down and make eye contact, and offer coffee and other nourishment. *The expression of feelings helps family caregivers to regain and maintain control of the situation.*

3. Provide truthful information and support for the family regarding the client’s specific illness or condition. *Attention needs to be given to methods of providing information and support.*

4. Assess for the influence of cultural beliefs, norms, and values on the families perception of coping. *What the family considers normal and abnormal coping behavior may be based on cultural perceptions.* (Ackley, Ludwig, 2008)

**Conclusion:**

*This patient’s prognosis is very poor, and this case is difficult to assess* due to the patient’s co-morbidities. The anoxic brain injury makes a neurologic assessment difficult. Treatment has been conservative and the results from the EGD will hopefully provide some answers and treatment options. Because of the patient’s age and health history, hemodialysis would not be ideal. The metabolic acidosis was resolving with the IV infusion of sodium bicarbonate, but would probably return if the chronic renal failure was not treated.

There would need to be some exploration of the patient’s family understanding of the disease process and what would be the patient’s wishes for end of life care. This would be a patient that would benefit from hospice and palliative care because resolution of the chronic
renal failure is unlikely. There needs to be some consideration of risks versus benefits of treatment.

The care this far has been to maintain oxygenation on the chronic ventilator, treatment of infections with antibiotic therapy, and an IV lasix infusion to minimize the edema associated with the renal failure.

The emphasis on the patient’s care has been to maintain oxygenation on the chronic ventilator, to treat infections with antibiotic therapy, and to use an IV lasix infusion to minimize the edema associated with renal failure. At the time of my clinical assessment, the patient’s status was classified as category one, meaning that doctors would resuscitate the patient if he were to code.

However, the doctors on my case were to meet with the patient’s family because they believed that his prognosis was so poor, his status should be changed to a do not resuscitate order.

Treatment of the patient’s renal failure would not benefit his long-term care.
References


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