

Patient Profile

On September seventh, 2010 a 77 year old white male presented to the emergency department with chief complaints of weakness, respiratory distress, and altered mental status. The patient is single and lives alone. He has only one living relative, a niece, who lives in Virginia. A caretaker, who checks on the patient every other day, brought him to the hospital after she found him on the toilet too weak to move himself. The patient's weight upon admission was 104.7 kg with a height of five feet and ten inches. According to his nurse, he has a good appetite and was able to finish 75 to 100 percent of his meals with help feeding. Very little history could be obtained on the patient. Noted by the doctor as a poor historian with an altered mental status, the patient frequently fell asleep while being interviewed by his attending nurses and doctors. From his medical records the patient was found to have a history of prostate cancer and decubitus ulcers and his primary attending physician suspected a history of chronic kidney disease. The patient was initially admitted to the progressive care unit and was eventually found to have a total of eight decubitus ulcers of varying stages. On the patient's second day of stay, a consult with the resident nephrologist determined that the patient's renal failure was acute rather than chronic.

Disease Background

Acute respiratory distress syndrome (ARDS) can be described as lung injury that causes acute-onset arterial hypoxemia, which cannot be corrected by oxygen therapy alone and typically lasts from days to weeks (1). Mechanical ventilation is the core of treatment for ARDS in order to properly oxygenate the patient (1). Due to hypercatabolism, energy and protein requirements are elevated in ARDS; protein should be about 1.5-2 g per day to help restore nitrogen balance (2). However, it is imperative not to overfeed these patients and exceed the carbon dioxide capacity of the respiratory system, as this would exasperate the problem of weaning the patient from the ventilator (2).

Decubitus ulcers, also known as pressure ulcers, are inflammations, sores, or lesions of the skin over a bony prominence. They occur most commonly in the obese, chronically ill, elderly, infected, and malnourished patients (3). The most common recommended requirements for wound healing are 30 to 35kcal per kilogram and anywhere from 1.25 to 2g of protein per kilogram of body weight (4). Studies of enterally fed patients have supported the use of higher protein formulas, citing a much larger decrease in the size of the ulcer compared to a standard formula (4). Specific amino acids such as arginine and glutamine, along with the leucine metabolite B-hydroxy B-methylbutyrate, play a role in collagen and epithelial cell production and are commonly supplemented in wound patients for this reason (4). One study has shown that resting energy expenditure is positively correlated with the surface area of wounds; however, there are no guidelines for nutrient requirements based on size or number of wounds (4). Overfeeding should be avoided because hyperglycemia can increase the risk of infection in the healing wound (4). Daily multivitamins, vitamin C, vitamin A, and zinc are also supplemented for wound healing (4).

Acute renal failure (ARF), also known as acute kidney injury or renal insufficiency, is an abrupt and sustained reduction in renal function (5). Due to the marked decrease in glomerular filtration rate, waste and fluid buildup in the body leading to hyperkalemia and metabolic acidosis (6). ARF rarely

occurs without a co-existing disease process (6). According to Lameire and colleagues, anywhere from five to 20 percent of critical care patients experience ARF during the course of their illness (7). The most likely causes are sepsis and multi-organ dysfunction syndrome. The same septic factors which cause arterial vasodilation also cause renal vasoconstriction and hypoxia. This contributes to necrosis and the development of ARF (7). Other causes are hypovolemia related to congestive heart failure or decompensated cirrhosis (7).

ARF also induces a general hypercatabolic state with unique changes in the metabolism of all three macronutrients (6). These changes are not only influenced by ARF, but also by any accompanying disease process or organ dysfunction (6). A typical ARF patient will be in negative nitrogen balance (6). The acidotic state caused by ARF contributes to muscle protein catabolism (6). Also, amino acid(AA) uptake into muscle cells is decreased, in part due to insulin resistance caused by ARF, while AA uptake by hepatic cells increases; leading to increased production of glucose and urea (6). This increased glucose production and the aforementioned insulin resistance aggravate hyperglycemia in the ARF patient (6). Hyperlipidemia is also frequently encountered in ARF because lipolysis is impaired (6).

Renal cells can repair or regenerate with adequate reperfusion and treatment of the underlying disease (7). Typical medical treatments which focus on reperfusion include administration of diuretics, volume therapy, and dopamine as a vasodilator (7). Renal replacement therapy is often needed to prevent complications from electrolyte abnormalities, acid/base imbalance, and fluid overload such as cardiac compromise and pulmonary edema (7). Prognosis for ARF patients is poor with a 50 to 80 percent mortality rate (8). The risk of mortality is strongly correlated with malnutrition, as defined by a pre-albumin less than 11 mg/dl, regardless of existing co-morbidities or severity of ARF(9).

Valencia and colleagues suggest using the Risk, Injury, Failure, Loss, and End-stage kidney criteria (RIFLE) for determining ARF patient's protein needs individually (5). With the RIFLE model, needs are based on the amount of protein catabolism in each patient, determined by the Urea Nitrogen Appearance rate (UNA). Minimally catabolic patients with less than 6g of UNA above their dietary intake should be limited to 0.6 to 0.8 g/kg of protein. This restriction serves to prevent the need for dialysis but should not be used long term (5). A UNA of 6 to 12g above intake is seen in moderately hypercatabolic patients with coexisting infections and injury; these patients will need 0.8 to 1.2g/kg (5). A UNA greater than 12g above intake is typically seen with severe underlying disease, dialysis, infection or trauma and may require 1.2 to 1.5 g/kg; malnutrition will further increase protein needs (5). However, according to the National Kidney Foundation amounts greater than 1.7g/kg will not improve nitrogen balance and 1.5 g/kg should be used as the upper limit (6).

Caution should also be used with energy, as expenditure is usually not more than 130 percent of normal for the ARF patient (6). The European Society for clinical Nutrition and Metabolism recommend 20 to 30kcal/kg for enterally fed ARF patients (10). Along those same lines, the National Kidney Foundation suggests a baseline of 25-30kcal/kg should be used with 35kcal/kg as the upper limit; complications from overfeeding outweigh the risks of underfeeding (6).

Determining the correct nutrition therapy in a patient with multiple disease processes requires a delicate balance. For example, vitamin C is metabolized to oxalic acid and in a healthy kidney even excessive amounts of vitamin C are cleared efficiently (11). However, during kidney failure oxalate builds up in the renal tubules, further impairs function, and delays recovery (11). This contraindicates the use of vitamin C for wound healing in an ARF patient. Electrolyte disorders are frequent in ARF patients (12) so it is important for the dietitian to be mindful of their concentrations in nutrition supplements and enteral formulas that may be commonly used for coexisting morbidities such as wounds or respiratory failure.

Current Admission

This patient's case involved many concurrent disease processes and complications, making it necessary for multiple diagnostic procedures and therapies to be utilized throughout the hospital course as numerous diagnoses were explored. The patient's first diagnosis of pneumonia with secondary hypoxia was the initial focus of treatment. This included chest x-rays, which revealed right upper and lower lobe hazy infiltrates, and antibiotic therapy using Levofloxacin. The patient was tachycardic for which he was monitored on telemetry. And with elevated levels of white blood cells and cardiac troponin, a possible myocardial infarction was explored. For this an echocardiogram was performed which was grossly normal; showing a mildly enlarged left atrium and grade I diastolic dysfunction, also known as a relaxation abnormality. Due to abnormal renal function labs, a diagnosis of acute renal failure was given. Doctor's notes attributed the ARF to sepsis and intravascular volume depletion or the pneumonia. A Foley catheter was placed and lasix was administered. While in the hospital the patient's respiratory distress progressed into hypercapnic respiratory failure, leading to endotracheal intubation. A dobhoff tube was placed at this time for nasogastric feedings. A Quinton catheter was placed a few days after intubation in anticipation of hemodialysis. Over the course of the patient's stay he was diagnosed with a total of eight pressure ulcers including a full thickness eschar on the buttocks in the shape of a toilet seat. After many failed attempts to wean the patient from the ventilator, due to excessive mucociliary secretions, a percutaneous tracheostomy was performed on day eight of stay.

Throughout the patient's stay he was treated with multiple antibiotics including Levofloxacin, Linezolid, Vancomycin, Meropenem; all of which cause diarrhea. Levofloxacin cannot be taken with orange juice, antacids, magnesium, calcium, Iron, Zinc or enteral formula; these items must be consumed 2 hours prior or 2 hours after the medication. To counteract the diarrheal side effects of the antibiotics, a probiotic, Lactobacillus was used.

Furosemide, a diuretic used to treat the edema associated with kidney disease may cause anorexia, thirst, cramps, nausea and vomiting, diarrhea, constipation and hyperglycemia. The patient was also on a variety of antihypertensive medications throughout his stay including Amlodipine, Hydralazine, Metoprolol, and Lisinopril; all of which require avoidance of natural licorice. Anorexia may be caused by Lisinopril and Hydralazine. Diarrhea may be caused by Hydralazine and Metoprolol. Food decreases the availability of Hydralazine, whereas food increases the availability of Metoprolol. Anti-

gerd medication Nexium should be taken 30-60 minutes before a meal with an acidic juice, it may decrease the absorption of Iron and B12. Antifungal medication Fluconazole has many nutritional side effects including taste changes, dry mouth, dyspepsia, nausea and vomiting, and diarrhea. Finally, Risperidone, an antipsychotic, increases appetite and weight gain and also causes constipation.

Nutrition Care Process

At admission the patient weighed 104.7 kg and is five feet and ten inches tall, putting him at an obese BMI of 33kg/M². Upon the initial nutrition consult the patient was on a renal diet, of which he was able to consume 75 to 100 percent of his meals with the help of his nurse. At this time the patient's GFR was 33 with elevated levels of creatinine and blood urea nitrogen (BUN), at 1.97mg/dl and 85mg/dl respectively. At the initial assessment the pt was believed to have Chronic Kidney disease (CKD) and, while the presence of wounds had been noted on the chart, the number and severity had not. Given this information, the patient's protein needs were estimated to be 0.8- 1.2 grams per kilogram of adjusted body weight or 66-100 grams per day, using the upper end of CKD recommendations to the lower end for wound healing. The patient's energy needs were estimated at 30kcal per Kg of adjusted body weight or about 2500kcal per day. Given the medical diagnosis, the patient was classified as severely compromised. The nutrition diagnosis at this time was increased nutrient needs related to wound healing and chronic kidney disease as evidenced by GFR 33 and unstageable pressure ulcer. The patient was also found to have self feeding difficulty related to altered mental status as evidenced by need for nurse assistance. Two interventions, changing the diet consistency to mechanical soft to aid in his feeding, and sending a Nepro twice daily to supplement the 60 grams of protein supplied by his renal diet with an additional 40 grams of protein, were initiated. A renal multivitamin was also recommended in a note to the doctor.

On the second day of stay the patient was transferred to the ICU where he was intubated and started on Pulmocare at 60ml per hour. It was now determined that the renal failure was acute rather than chronic. At this time his energy needs were reassessed, using the Ireton-Jones ventilator specific equation, to be 1970Kcal. His prealbumin was taken with the daily labs and found to be very low at 9mg/dl. Given this malnutrition, his protein needs were now determined to be higher at 1 to 1.3g/kg of adjusted body weight, or 85-110g per day. The Pulmocare was providing 2160Kcal and 90g of protein. The patient's GFR was continuing to fall as his BUN and creatinine continued to rise; in anticipation of dialysis a catheter was placed. We recommended continuing the current tube feeding, and monitoring the patient's tolerance and lab values until dialyzed, when we would recommend a more appropriate formula.

By the third day of stay the patient's labs showed signs of recovering renal function and plans for dialysis were put on hold. On day six GFR was back to 51, BUN and Creatinine had decreased significantly to 48mg/dl and 1.35mg/dl respectively. Official documentation from the wound care nurse now listed eight different pressure ulcers of all stages. One 30ml packet of Prostat was ordered to be administered daily with tube feedings, providing an additional 15g of protein, or 1.27 g/kg of adjusted body weight. The patient was extubated and transferred to PCU briefly, returning to the ICU with recurring respiratory symptoms.

On return to the ICU, day of stay 10, Prealbumin had increased to 11 mg/dl. With renal function almost returned to normal, the goal was to continue to increase protein intake to bring prealbumin back to normal and encourage wound healing. In a note to the doctor I recommended increasing Prostat to T1D; these recommendations were not followed by the doctor.

Over the course of the next seven days the patient began to lose renal function again. Due to the patient's altered mental status I never spoke to him directly. However, I did speak to his family member to find that discharge planning had already begun. The patient and his family member decided on palliative care rather than long term acute care. Swallow evaluations had been performed and the patient was at high risk for aspiration. Despite this, the patient opted to discontinue tube feedings in favor of pleasure feedings and was discharged to hospice on day 16 of his hospital stay.

Summary/Conclusion

This patient's presentation of acute renal failure was not atypical; its etiology multi-factorial, with both common co-morbidities such as sepsis and other aggravating and competing metabolic demands, such as wound healing. While the patient did show signs of recovery, unfortunately, as is predicted for most ARF patients, the outcome was poor. This was likely influenced by pre-existing malnutrition and the multitude of organ systems affected in this particular patient.

In a multi-factorial disease, such as ARF, nutrition care must be carefully tailored to the individual and the degree of metabolic changes, as these can vary greatly. Clinical judgment is critical; however tools such as the RIFLE criteria can be very helpful in choosing the correct nutrition therapy. Knowledge of the specific metabolic changes which cause hypercatabolism in ARF, specifically how protein use is impaired, is also important. This can help the practitioner understand the limits of nutrition therapy in actually affecting clinical outcomes, such as nitrogen balance. And may also help guide them in using biochemical markers, such as protein stores, for determining when to progress or change the course of nutrition therapy.

There were slight differences in the nutrition recommendations for the three diseases focused on in this paper: ARF, ARDS, and decubitus ulcers; however, all cause at least slightly increased needs and all have risks associated with overfeeding which can negatively impact the healing process. For us, the most difficult decisions to make came from balancing our perceived extreme increase in the patient's metabolic needs for wound healing, up to 35 Calories and two grams of protein per kilogram, with the limits of CO₂ capacity, impacting the patient's ability to recover from respiratory distress and wean from the ventilator. It was also difficult to decide whether our recommendations should be more focused on preserving the limited function of the kidney or addressing the high protein needs for wound healing.

After having researched these diseases more carefully myself I do not believe I would make many changes to our nutrition care process. Some may argue that the patient should have been treated more aggressively at the first consult for the wounds; that wounds are a bigger threat to the patient's

health and that if kidney function were to worsen as a result, that dialysis is available. However, the extent and number of wounds was not known at this time and given the patient's poor diet prior to admission, a slow and cautious increase in protein ensured that complications would not occur. I still debate as to whether we should have recommended an enteral formula specific to kidney disease over the Pulmocare which the doctor had ordered. The macronutrient distribution in this feeding was roughly similar to our estimates, however, electrolytes and micronutrients were not tailored to the ARF, and as a result potassium and phosphorous labs exceeded normal limits at times during this patient's stay. Further research and discussion among practitioners is needed to help solve these and other dilemmas in difficult case studies such as this.

Acute Renal Failure and other Co-Morbidities

References

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