

Sara E. Levy

Pathophysiology Term Paper

Long Island University

Pathophysiology Term Paper

1. The systemic effects of dehydration can be far reaching and permanently damaging if not dealt with appropriately in a timely manner. An immediate systemic result of dehydration is a decrease in blood volume. This decrease in blood volume can lead to Pre-renal Failure which is a classification of Acute Renal Failure (ARF). As stated in Essentials of Pathophysiology, "Renal failure is a condition in which the kidneys fail to remove metabolic end products from the blood and regulate the fluid, electrolyte, and pH balance of the extra-cellular fluids. (Porth, 2011 pp. 641)" Acute renal failure refers to renal failure which is sudden in onset and can be reversed if treatable factors are recognized early and treated appropriately. Pre-renal failure in particular is marked by a decrease in renal blood flow, a direct consequence of dehydration. Under normal conditions the Kidneys receive as much as 22% of blood flow, a rate which is sufficient to facilitate the filtration of metabolic wastes and allows for body fluid and electrolyte regulation. (Porth, 2011)

2. Generally, the kidneys are able to sustain a relatively large decrease in blood flow before ischemic damage occurs. However, as renal blood flow falls, the glomerular filtration rate (GFR) also drops leading to the two main indicators of pre-renal failure, oliguria and a disproportionate rise in BUN levels in proportion to creatinine levels. (Porth, 2011)

A decrease in glomerular filtration rate (GFR) leads to oliguria, low urine output. Therefore an early sign of acute renal failure is a sharp decrease in urine output. Furthermore, a low fractional excretion of sodium suggests oliguria due to specifically to decreased renal perfusion, which is a direct result of low blood volume. The nephrons respond to the low blood volume levels by decreasing urine output in an attempt to maintain vascular volume and prevent ischemic changes. (Porth, 2011)

Furthermore, a drop in GFR creates a disproportionate ratio of BUN to creatinine levels. A normal BUN to creatinine ratio is 10:1. However, an abnormal ratio is the result of creatinine being excreted in urine while urea is reabsorbed into the blood stream. Since creatinine is a large molecule it

is non-diffusible during periods of decreased GFR, and will therefore be excreted with the urine since it cannot be reabsorbed into the blood. By comparison, small particles such as urea will be reabsorbed the blood in situations of GFR into which leads to a raised BUN level. Consequently, a situation arises where the BUN levels will be greatly disproportionate in comparison to creatinine, with a general ratio greater than 20:1. (Porth, 2011) Indeed, this is reflected in lab results for the scenario presented. The patient has a BUN of 60 mg/dL and a creatinine of 1.2 mg/dL. While creatinine levels fall within a normal range, the BUN is almost three times larger than the accepted range.¹

3. Prolonged pre-renal failure will eventually lead to intrinsic renal failure. Intrinsic renal failure, also known as intrarenal failure, is characterized by prolonged ischemic changes in renal tubular cells due to a lack of blood flow which lead to Acute Tubular Necrosis (ATN). ATN is specifically characterized by the destruction of epithelial tissue in the renal tubules in conjunction with suppression of renal functioning. When blood flow falls to 25% or less of the regular volume, ischemic changes occur, and tubular epithelial cells are most sensitive to ischemic changes due to their high metabolic rates. Renal tubular ischemia causes epithelial tissue sloughing and necrosis which leads to obstruction of renal tubules and increased intraluminal pressure which decreases glomerular filtration. What is more, a decrease in glomerular capillary filtration pressure, caused in part by tubuloglomerular feedback mechanisms, is the result of afferent arteriolar constriction. The tubular injury and increased luminal pressure causes fluid to move into the interstitium from the tubular lumen, which can give rise to edema. (Porth, 2011)

4.a. The pathogenesis of ATN can be divided into three clinical stages: onset, maintenance, and recovery. ATN begins with the onset or initiating phase, which can last hours or days, and begins at the precipitating event, such as the ischemic phase of prerenal failure, up to the time that tubular injury takes place. (Porth, 2011)

¹ Creatinine 0.5-1.4 mg/dL; Urea Nitrogen (BUN) 7-21 mg/dL (W.B.Saunders Company, 1999)

The second stage, known as the maintenance phase of ATN, is marked by a decrease in GFR which causes sudden retention of endogenous metabolites that would normally be cleared by the kidneys, such as urea, potassium, sulfate, and creatinine. In particular, the sloughing and necrosis of tubular cells lead to obstructions in the renal tubules and subsequently increased intraluminal pressure. The tubular injury and increased luminal pressure causes fluid to move into the interstitium from the tubular lumen, causing edema. Furthermore, aside from edema, the overall reduction in glomerular filtration leads to fluid retention which gives rise to water intoxication and pulmonary congestion, while oliguria (low urine output) reaches its lowest point. (Porth, 2011)

Lastly, the recovery phase refers specifically to the time when renal tissue is repaired. Signs that tissue repair is well underway include a gradual increase in urine output and a fall in serum creatinine. It is worth noting that diuresis is common before renal function has completely returned to normal. In some cases diuresis can be severe enough to cause excessive loss of water and electrolytes while BUN and serum creatinine levels, potassium and phosphate levels remain elevated or continue to rise despite the increased urine output. In time, improvement in urine concentration takes place once renal tubule function is restored, though mild to moderate kidney damage may persist in some cases. (Porth, 2011)

4.b. Nursing interventions can take different forms when attempting to prevent ATN due to prerenal failure particularly in relation to dehydration. At an early stage, ATN can certainly be prevented from reaching the initiating stage by the Nurse teaching the patient to increase and monitor the intake of fluids to avoid dehydration. Should initial signs of dehydration be observed, the Nurse should intervene by ensuring that the patient drinks plenty of water, and if need be, consult a Dr. and have an IV ordered to hydrate the patient. (DOI: Evolve Elsevier Care Plan Constructor)

If a patient were to present in a hospital setting already suffering from the second, maintenance, phase of ATN, the Nurse needs to evaluate the defining characteristics of the patient's ARF due to ATN and plan interventions accordingly. As previously mentioned in this paper, a patient

could suffer from various symptoms including though not limited to azotemia, blood pressure changes, edema, oliguria, pulmonary congestion, and specific gravity changes. In the case presented, amongst other problems, the patient is clearly suffering from azotemia and blood pressure changes as a result of dehydration and the resultant ATN. A nursing intervention for azotemia would be to monitor serum and urine osmolality and BUN/creatinine ratio. These measures of concentration will be altered with increased fluid volume and eventually will indicate repair in renal tubules. A nursing intervention related to the patient's decreased blood pressure would be to monitor for side effects of diuretic therapy. In particular orthostatic hypotension (especially if the client is also receiving ACE inhibitors), hypovolemia and electrolyte imbalances (hypokalemia and hyponatremia) are of concern. This is of particular importance considering the patient presented with a very low blood pressure. (DOI: Evolve Elsevier Care Plan Constructor)

Lastly, during the recovery phase, nursing interventions include the careful maintenance of the rate of all IV infusions, carefully utilizing an IV pump to facilitate the fluid intake of the patient and once again monitoring serum and urine osmolality, and BUN/creatinine ratio. As previously mentioned, lab abnormalities will return to normal once the increased fluid volume enables the renal tubules to return to a normal functioning level; therefore monitoring IV infusions and I&O charts in conjunction with lab values indicate that the renal tubules are repairing properly. (DOI: Evolve Elsevier Care Plan Constructor)

References

Porth, C. M. (2011). *Essentials of pathophysiology*. (3rd ed., pp. 641-4). Philadelphia: Wolters Kluwer Health; Lippincott Williams & Wilkins.

W.B.Saunders Company. (1999). *Normal reference range table*. Retrieved from
<http://pathcuric1.swmed.edu/pathdemo/nrrt.htm>

DOI: Evolve Elsevier Care Plan Constructor. Retrieved from
<http://evolvels.elsevier.com/section/content/Default.asp>