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Pathophysiology of Sepsis-Associated Acute Kidney Injury

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Introduction.

• 40% of critical care patients have AKI and about half of those patients are also suffering from sepsis (Pettia & Bellomo, 2014).

• As a nurse on a progressive care medical oncology unit, sepsis patients are encountered on a daily basis and many of them are also diagnosed with AKI.

• Through research, the author hopes to gain a better understanding of the connection between AKI and sepsis.

• As a master’s student studying to be a nurse practitioner, the author hopes to gain knowledge that can be applied to future practice.

Underlying Pathophysiology and Significance of Pathophysiology

Septic associated acute kidney injury involves alterations in microcirculation and impairment of normal hemostasis. According to Dirkes (2013) the initial injury causes endothelium to leak protein-rich fluid into the subcutaneous tissues, which then initiates an inflammatory response. Neutrophils respond and adhere to the endothelial cells in the injured area. The body activates cachectolamines, which cause vasocostriction and vasodilation. Amount of perfused capillaries are reduced and vessels become clogged with neutrophils. This causes a cessation of blood flow and in turn leads to hypoxia of tissue (p. 126). According to Shum, Yan, and Chan (2015) when the vessels are clogged it takes a long time for leukocytes to pass through which results in a longer exposure time of the endothelium to cytokine, and pathogens which then triggers more inflammatory signals and leads to more oxidative stress. According to Dirkes (2013) this inflammation and endothelial injury leads to activation of coagulant system and cause a cycle of vascular injury and cell death. It has been discovered that when microcirculation is rapidly improved there is an increased chance for sepsis survival and improvement in organ function within 24 hours (p. 126). Venkatachalam and Weinberg (2012) describe this release of cytokines as a “cytokine storm” accompanied by peripheral vascular resistance and hypotension (p. 81). According to Alobaidi, Basu, Goldstein, and Bagshaw (2015) “cellular hypoxia is a molecular driver of injury during SA-AKI. Tissue hypoxia in the kidney during sepsis may be defined by inflammation, changes in intrarenal nitric oxide, nitrosative stress, or oxygen radical homeostasis and dysregulation (p. 6). (Enzo Life Sciences, 2014)

According to Dirkes (2013) the septic response impairs homeostasis. Inflammation of the microcirculation is what alters homeostasis. The exposure of the endothelium to cytokines results in alterations of function. This can cause abnormal balance between vasoactive compounds (nitric oxide, prostacyclin), which leads to loss of vascular tone and microvascular perfusion heterogeneity. Circulatory disruption occurs including cellular debris occluding the vessel, which leads to production of cytokines and reactive oxygen species and causes an imbalance between procoagulant and anticoagulant mechanisms (p. 127).

According to Dirkes (2013) In the kidney, proinflammatory cytokines, tumor necrosis factor-alpha, and Interleukin-6 are generated by injured renal tubule cells or from extracellular areas and are a contributor to renal injury. The influx of all of the inflammatory cytokines leads to congestion and therefore slows renal blood flow leading to AKI. If hyperperfusion of renal system persists, AKI progresses to structural tubular injury (p. 127). Another study done by Xu, Chang, Hack, Eadon, Alper, and Cunningham (2013) looks at the pathological changes of the glomerular endothelium and found that tumor necrosis alpha plays a huge role in these changes (p. 79).

New Research

• Clinical trials based on molecular approaches have poor results.

• Current therapy is aimed at management of hemodynamics including administration of crystalloids (Schortgen & Asfar, 2015).

• Albumin is a second line therapy. (Schortgen & Asfar, 2015).

• Recommendations are to give fluid to maintain a mean arterial pressure (MAP) of 65-70. Trials keeping MAP 80-85 showed no decrease in mortality from trials keeping MAP 65-70 (Schortgen & Asfar, 2015).

• Early administration of antibiotics is associated with increased survival rates. (Schortgen & Asfar, 2015).

• The alkaline phosphate is thought to neutralize bacterial endotoxins and catalyze the conversion of adenosine triphosphate into adenosine, a potent anti-inflammatory factor. This reduces inflammation and leads to decreased sepsis associated acute kidney injury (Swaminathan, Rosner, & Okusa, 2015).

• Alkaline phosphate administration phase 2a trials have been shown to reduce sepsis associated AKI (Swaminathan, Rosner, & Okusa, 2015).

Implications for Nursing Care

• There is still much to be discovered about sepsis associated acute kidney injury.

• Implications for nursing include recognition of SIRS criteria and initiation of current treatments including early antibiotic administration and fluid resuscitation.

• Since mortality is such an issue with sepsis associated AKI, many clinical trials are currently underway which are sure to bring about upcoming changes in clinical practice.

• It is important for nurses of all levels to stay up to date on current research and practice related to this currently prevalent critical care issue.

Conclusion

• Sepsis is a common cause of AKI.

• The research shows that AKI associated sepsis has high morbidity and mortality rates.

• Even if a patient survives the acute phase of kidney injury there are many chronic consequences that can occur as a result.

• This makes keeping up with further research and developments related to AKI the more important to nurses.

Additional Sources