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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 (Pettila & Bellomo, 2014).

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 catecholamines, which cause vasoconstriction and vasodilation. Amount of perfused capillaries is reduced and veins 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 venules 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 oxidant stress. According to Dirkes (2013) this inflammation and endothelial injury leads to activation of coagulant system and causes a cycle of vascular injury and cell death. It has been discovered than 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 Alboabdi, 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).

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 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 change in clinical practice. It is important for nurses of all levels to stay up to date on current research and practice related to this extremely 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 all the more important to nurses.

References

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Additional Sources