Journal of Medicine and Medical Sciences Vol. 14(1) pp. 1-3, January, 2023
Available online https://www.interesjournals.org/medicine-medical-sciences.html

Review Article

Infectious Diseases and Systemic Sepsis and Septic Shock

Andriole Katherine*

Brigham and Women's Hospital, Harvard Medical School, Department of Radiology, Boston, Massachusetts, United States

*Corresponding Author's E-mail: dravrah.hwb.andriole@eloirdnak.edu

Received: 02-Jan-2023; Manuscript No: jmms-23-87046; **Editor assigned:** 3-Jan-2023; Pre-QC No: jmms-23-87046 (PQ); **Reviewed:** 16-Jan-2023; QC No: jmms-23-87046; **Revised:** 23-Jan-2023; Manuscript No: jmms-23-87046 (R); **Published:** 30-Jan-2023, DOI: 10.14303/2141-9477.2023.18

Abstract

Introduction: Numerous bacterial, viral, fungal, and parasitic diseases involve the kidney. The majority of systemic infections do not involve the kidneys at all; however, in some cases, renal failure may be the presenting symptom and the most significant challenge in treatment. A purely anatomic approach to the classification of infectious diseases affecting the kidney is rarely helpful because the majority of infections may involve several different aspects of renal function. However, individual infectious processes may have a tendency to involve the renal vasculature, glomeruli, interstitium, or collecting systems.

Vibrio cholerae causes gastroenteritis, or cholera. Vomiting, severe secretory diarrhoea, and dehydration are the symptoms. Acute tubular necrosis as a result of dehydration or the infection itself can result in severe complications, including electrolyte imbalances and oligoanuric acute kidney injury. However, no cases of cholera with severe proteinuria and acute kidney damage have been reported. This study was therefore conducted. Goals and Intentions The purpose of this study was to evaluate the clinical features, treatment, and prognosis of Acute Kidney Injury (AKI) in cholera patients. to link proteinuria and Acute Kidney Injury (AKI) in cholera patients; and to contrast patients with AKI and those with normal kidney function who have cholera.

Methods and Materials: Cholera patients were the subjects of this retrospective observational study. Amount of proteinuria, acute kidney injury, and prognosis in cholera patients were gathered. The majority of patients suffered from hypovolemic shock, severe to severe diarrhea, dehydration, and significant vomiting.

Keywords: Nephrotic Syndrome, Severe Acute Respiratory Syndrome, Disseminate Intravascular Coagulation, Hemolytic Uremic Syndrome, Hemorrhagic Fever

INTRODUCTION

Numerous bacterial, viral, fungal, and parasitic diseases involve the kidney. The majority of systemic infections do not involve the kidneys at all however, in some cases, renal failure may be the presenting symptom and the most significant challenge in treatment. A purely anatomic approach to the classification of infectious diseases affecting the kidney is rarely helpful because the majority of infections may involve several different aspects of renal function. However, individual infectious processes may have a tendency to involve the renal vasculature, glomeruli, interstitium, or collecting systems. A microbiologic classification of the organisms that affect the kidney is used in this chapter. Although urinary tract infections and Haemolytic Uremic Syndrome (HUS) are important causes of renal dysfunction in infectious diseases, they are not discussed in depth because they are considered separately (Albert Mathieu et al., 2007).

The geographical distribution of infectious diseases in various nations must be carefully taken into consideration when determining the cause of renal involvement in a child with evidence of infection. A background of traveling abroad; exposure to insects, animals, and unusual foods and beverages, activities in nature, such as hiking or swimming; every time, contact with infectious diseases must be avoided. The skin and mucous membranes should be carefully examined during the clinical examination, and insect bites, lymphadenopathy, and involvement of other organs should be checked for. The underlying infection will be easier to diagnose and treat if you work closely with a hospital microbiologist and a pediatric infectious disease specialist. *Vibrio cholerae* causes gastroenteritis, or cholera. Vomiting, severe secretory diarrhoea, and dehydration are the symptoms. It looks like rice-water-like stools with a fishy odor. It can result in severe complications, including Oligoanuric Acute Kidney Injury (AKI) due to Acute Tubular Necrosis (ATN) and severe electrolyte imbalances like hyponatraemia, hypernatremia, and hypokalaemia. Dehydration or infection itself can lead to renal complications like AKI, metabolic acidosis, ATN, and Acute Tubulo Interstitial Nephritis (ATIN). However, no cases of cholera with severe proteinuria and acute kidney damage have been reported. Even regarding cholera presenting with AKI, there is a dearth of data. This study was therefore conducted (Anspach Renee R, 1988).

DISCUSSION

Systemic Sepsis and Septic Shock

In systemic sepsis, impairment of renal function is common. The degree of involvement in the kidney can range from mild proteinuria to acute renal failure necessitating dialysis, depending on the severity of the infection and the organism responsible. As part of systemic sepsis, the organisms that cause acute renal failure vary based on age and location, as well as between healthy and immunocompromised children. Group B *Streptococci*, coliforms, *Staphylococcus aureus*, and *Listeria monocytogenes* are typically to blame during the newborn period. The majority of infections in older children are caused by *Neisseria meningitides*, *Streptococcus pneumonia*, and *S. aurous*. A wide variety of bacteria are found in immunocompromised individuals, and additional pathogens, such as *Haemophilic influenza*, *Salmonella* species, and *Pseudomonas pseudomallei*, must be considered in tropical countries (Bassett Andrew Mark et al., 2018).

However, since the introduction of the H. influenza type B vaccine, the prevalence of severe systemic infections caused by this organism has significantly decreased. Nonspecific symptoms are typically seen in systemic sepsis: tachycardia, fever, tachypnea, and evidence of skin and organ perfusion Multifactorial factors play a role in the renal involvement in systemic sepsis' pathophysiology. The first sign of hypovolemia is decreased renal perfusion, which is caused by the loss of plasma from the intravascular space and increased vascular permeability. Due to the depressant effects of endotoxin or other toxins on myocardial function, hypovolemia frequently occurs alongside depressed myocardial function. Oliguria is a common and early sign of severe sepsis because the renal vasoconstrictor response to decreased circulating volume and decreased cardiac output further reduces glomerular filtration. In sepsis, a number of vasodilator pathways are activated, including the Kinin and Nitric Oxide pathways. Vascular beds may dilate improperly as a result. Adults with sepsis caused by Gram-negative organisms frequently experience vasodilation of the capillary beds, which can result in warm shock. Children, on the other hand, typically experience intense vasoconstriction as the typical response to sepsis (Beagan Brenda L, 2000).

Reversible perennial failure is followed by established renal failure with the characteristics of vasomotor nephropathy or acute tubular necrosis if renal under perfusion and vasoconstriction are severe and persistent. Direct kidney damage caused by endotoxin and other toxins, as well as the release of inflammatory mediators like Tumor Necrosis Factor (TNF) and other cytokines, arachidonic acid metabolites, and proteolytic enzymes, are additional mechanisms of renal damage in systemic sepsis. It is hypothesized that Nitric Oxide (NO) plays a significant role in the pathophysiology of renal failure in sepsis. It is still unknown whether increased NO has beneficial or detrimental effects on the kidneys. Saline resuscitation was found to be superior in trials of selective NO synthetase inhibition. Because it maintains renal blood flow and glomerular filtration, NO may be beneficial in endotoxemia. Coagulation activation may play a role in renal impairment and is an important part of the pathophysiology of septic shock. In addition to downregulating antithrombotic mechanisms like the protein C pathway, there is activation of multiple prothrombotic and antifibrinolytic pathways. Activated protein C treatment has been shown to improve outcomes in adult septic shock, but it has not been shown to help children with sepsis and may increase the risk of bleeding, especially in infants (Beagan Brenda, 2003).

Mycobacterium tuberculosis

Mycobacterium tuberculosis is escalating worldwide. The spread of the Human Immunodeficiency Virus (HIV) infection epidemic, large influxes of immigrants from countries where Tuberculosis (TB) is common, the emergence of multiple-drug-resistant M. tuberculosis, and the breakdown of health services in various countries for effective TB control have all contributed to this rise. One-third of the world's population is currently infected with the TB bacillus, according to most estimates. Each year, approximately 2 million people die from more than 8 million cases of tuberculosis. In addition, 5-10 percent of people infected with the TB bacillus either develop the disease or contract it at some point in their lives. During the lymphohematogenous phase of childhood

TB, mycobacteria are widely distributed to numerous body organs following a respiratory illness in children. In many cases of miliary TB, tubercle bacilli can be found in the urine. In the glomeruli, hematogenously dispersed tuberculomata result in caseating, sloughing lesions that release bacilli into the tubules. Most of the time, the renal lesions don't cause any symptoms and show up as sterile pyuria or mycobacteria in the urine (Bell Ann V et al., 2014).

Infectious organisms can be discharged into the tubules, urethra, and bladder by rupture of tuberculomata in the cortex, which can either calcify and cavitate or rupture into the pelvis. Dysuria, loin pain, hematuria, and pyuria are the symptoms of this complication. However, even when radiologic and pathologic abnormalities are very extensive, the renal involvement is frequently asymptomatic. Cystitis with frequent urination and, in later cases, a contracted bladder may result from persistent *Tuberculous bacilluria*. In the majority of cases, the intravenous urogram is abnormal. Pyelonephritis with calyceal blunting and calyceal-interstitial reflux are the initial findings. Papillary necrosis is indicated by the appearance of papillary cavities later. Hydronephrosis, focal calcification, cavitation, and ureteric strictures are also possible. Typically, renal function is maintained well, and hypertension is uncommon. Interstitial nephritis is a sign of renal failure in some cases caused by the infection itself or reactions to chemotherapeutic agents (Betancourt Joseph R, 2006).

Classic symptomatic renal tuberculosis is a late and uncommon complication in children, occurring rarely less than four or five years after the initial infection. As a result, it is most frequently diagnosed in adolescents. Adult studies have shown that 6-10% of screened sputum-positive pulmonary TB patients have renal involvement, and that 26-75% of renal TB patients also have active pulmonary TB. Mycobacteria isolation from the urine or the presence of typical clinical and radiographic features in a child who has or has had TB are the two methods used to make the diagnosis. Isoniazid, rifampicin, pyrazinamide, and ethambutol are first given to treat renal TB for two months. Isoniazid and rifampicin are then given for another seven to ten months. These drug regimens are similar to those used to treat other types of TB. Ultrasonography or an intravenous program should be used to monitor patients with extensive renal involvement for late scarring and urinary obstruction (Bleakley Alan, Brice Julie, 2008).

CONCLUSION

AKI is the most common complication of cholera. Surprisingly, there was no significant link found between AKI and common risk factors like hypovolemic shock and dehydration. Possibly, our tertiary care hospital treated all of these risk factors appropriately, promptly, and promptly. However, proteinuria was found to have influenced the onset of AKI but had no effect on recovery. It may suggest that the recovery of AKI was influenced by other factors, such as the resolution of dehydration and the hypovolemic shock. This study suggests that *V. cholerae* infection rarely results in proteinuria and Acute Kidney Injury (AKI) with spontaneous kidney disease remission when appropriate treatment is administered (Bochatay Naïke, Bajwa Nadia M, 2020) (Braun Lundy et al., 2017).

ACKNOWLEDGEMENT

None

CONFLICT OF INTEREST

None

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