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Gas-Producing Vibrio Cholerae: A Case Report of Gastroenteritis with Acute Kidney Injury

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Introduction

Acute Kidney Injury (AKI), previously called acute renal failure (ARF), is a sudden decrease in kidney function that develops within 7 days, as shown by an increase in serum creatinine or a decrease in urine output, or both. Causes of AKI are classified as either prerenal (due to decreased blood flow to the kidney), intrinsic renal (due to damage to the kidney itself), or post renal (due to blockage of urine flow). Prerenal causes of AKI include sepsis, dehydration, excessive blood loss, cardiogenic shock, heart failure, cirrhosis, and certain medications like ACE inhibitors or NSAIDs. Intrinsic renal causes of AKI include glomerulonephritis, lupus nephritis, acute tubular necrosis, certain antibiotics, chemotherapeutic agents, and contrast dye used for imaging. Postrenal causes of AKI include kidney stones, bladder cancer, neurogenic bladder, enlargement of the prostate, narrowing of the urethra, and certain medications like anticholinergics. The diagnosis of AKI is made based on a person's signs and symptoms, along with lab tests for serum creatinine and measurement of urine output. Other tests include urine microscopy and urine electrolytes. Renal ultrasound can be obtained when a post renal cause is suspected. A kidney biopsy may be obtained when intrinsic renal AKI is suspected and the cause is unclear. AKI is seen in 10-15% of people admitted to the hospital and in more than 50% of people admitted to the intensive care unit (ICU). AKI may lead to a number of complications, including metabolic acidosis, high potassium levels, uremia, changes in body fluid balance, and effects on other organ systems, including death. People who have experienced AKI are at increased risk of developing chronic kidney disease in the future. Management includes treatment of the underlying cause and supportive care, such as renal replacement therapy. The clinical presentation is often dominated by the underlying cause. The various symptoms of acute kidney injury result from the various disturbances of kidney function that are associated with the disease. Accumulation of urea and other nitrogen-containing substances in the bloodstream lead to a number of symptoms, such as fatigue, loss of appetite, headache, nausea, and vomiting. Marked increases in the potassium level can lead to abnormal heart rhythms, which can be severe and life-threatening. Fluid balance is frequently affected, though blood pressure can be high, low, or normal.

Intrinsic or Renal

Intrinsic AKI refers to disease processes which directly damage the kidney itself. Intrinsic AKI can be due to one or more of the kidney's structures including the glomeruli, kidney tubules, or the interstitium. Common causes of each are glomerulonephritis, acute tubular necrosis (ATN), and acute interstitial nephritis (AIN), respectively. Other causes of intrinsic AKI are rhabdomyolysis and tumor lysis syndrome. Certain medication classes such as calcineurin inhibitors (e.g., tacrolimus) can also directly damage the tubular cells of the kidney and result in a form of intrinsic AKI. Postrenal AKI refers to acute kidney injury caused by disease states downstream of the kidney and most often occurs as a consequence of urinary tract obstruction. This may be related to benign prostatic hyperplasia, kidney stones, obstructed urinary catheter, bladder stones, or cancer of the bladder, ureters, or prostate.

Treatment

The management of AKI hinges on identification and treatment of the underlying cause. The main objectives of initial management are to prevent cardiovascular collapse and death and to call for specialist advice from a nephrologist. In addition to treatment of the underlying disorder, management of AKI routinely includes the avoidance of substances that are toxic to the kidneys, called nephrotoxins. These include NSAIDs such as ibuprofen or naproxen, iodinated contrasts such as those used for CT scans, many antibiotics such as gentamicin, and a range of other substances. Monitoring of kidney function, by serial serum creatinine measurements and monitoring of urine output, is routinely performed. In the hospital, insertion of urinary catheter helps monitor urine output and relieves possible bladder outlet obstruction, such as with an enlarged prostate. In prerenal AKI without fluid overload, administration of intravenous fluids is typically the first step to improving kidney function. Volume status may be monitored with the use of a central venous catheter to avoid over- or under-replacement of fluid. If low blood pressure persists despite providing a person with adequate amounts of intravenous fluid, medications that increase blood pressure

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(vasopressors) such as norepinephrine, and in certain circumstances medications that improve the heart's ability to pump (known as inotropes) such as dobutamine may be given to improve blood flow to the kidney. While a useful vasopressor, there is no evidence to suggest that dopamine is of any specific benefit and may in fact be harmful.

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