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Etiopathogenesis of acute kidney injury

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Acute kidney injury is defined by an abrupt decrease in kidney function that includes, but is not limited to acute renal failure. It is a broad clinical syndrome encompassing various etiologies, including specific kidney diseases like acute interstitial, glomerular or vasculitic renal diseases; non-specific conditions like ischemia, toxic injury or extra-renal pathology as pre-renal azotemia, or post-renal obstructive nephropathy. Acute kidney injury is common, harmful and potentially treatable. Even a minor acute reduction in kidney function has an adverse prognosis. Kidney failure is defined as a GFR of 15 ml/min per 1.73 m2 body surface area, or requirement for renal replacement therapy. Acute kidney injury complicates 5-7% of acute care hospital admissions and upto 30% of admissions at intensive care unit, particularly in the setting of diarrheal illness, infectious diseases like malaria, leptospirosis and natural disasters such as earth quakes. Mortality due to AKI may exceed 50% in cases admitted in intensive care unit. It also increases the risk for development or worsening of chronic kidney disease. Acute kidney injury may be community acquired or hospital acquired. Common causes of community acquired syndrome includes volume depletion, adverse effects of medication and obstruction of the urinary tract and those in hospital acquired AKI are sepsis, major surgical procedures, critical illness involving heart and liver failure, administrations of intravenous contrast and nephrotoxic medications. Pre-renal azotemia is the most common cause of acute kidney injury and usually a result of renal hypoperfusion. It accounts for approximately 60 to 70% of the community acquired and 40% of the hospital acquired cases. The most common clinical conditions associated with pre-renal azotemia are hypovolemia, decreased cardiac output, advanced cirrhosis and medications that interfere with renal autoregulatory responses such as NSAIDS and inhibitors of angiotensin II. Early in the course of pre-renal acute kidney injury, the renal parenchyma remains intact because kidney hypoperfusion initiates a neurohormonal cascade that results in afferent arteriolar dilatation like myogenic reflex, prostaglandin mediated and efferent arteriolar constriction, angiotensin II mediated thereby maintaining glomerular filtration pressure closer to normal and thus prevents marked reduction in GFR if renal blood flow reduction is not excessive. Intrinsic azotemia is classified according to the primary histologic site of injury. Renal tubular epithelial cell injury, commonly termed acute tubular necrosis, occurs more commonly in the setting of ischemia, although renal tubules can also be affected by sepsis and toxins both endogenous and exogenous.

Biography

Dwijen Das completed MBBS from Gauhati Medical College and MD (General Medicine) from Assam Medical College, Dibrugarh, Assam, India. He is currently working as an Associate Professor of Medicine and In-charge of Dialysis Unit, Silchar Medical College. He was immediate past Honorary General Secretary of API, Assam Chapter and a fellow of American College of Physicians. He has 20 publications to his credit in reputed indexed journals. He has contributed many chapters in Medicine updates published during Assam APICON and all India APICON. He is also an Assistant Editor of Assam Journal of Internal Medicine and peer reviewer of two journals of national repute and one international journal. He is the Editorial Board Member of the book, "Progress in Medicine" published in 2017.

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