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POSTOPERATIVE ACUTE RENAL FAILURE, THE ROLE OF ANESTHESIOLOGISTS.

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Acute renal failure can be defined as an abrupt decline in renal function with a decrease in glomerular filtration rate resulting in the retention of nitrogenous waste products. Acute renal failure has conventionally been classified as; Prerenal failure, denoting a disorder in the systemic circulation that causes renal hypo-perfusion, Intrinsic renal failure, where correction of the circulatory impairment does not restore the normal GFR. Intrinsic renal failure generally includes tubular necrosis. Postrenal obstructive failure is a third possibility[1].

Many studies defined the determinants of renal dysfunction including; serum creatinine, blood urea nitrogen, serum potassium, urine urea, creatinine clearance, urinary lysozyme, serum lysozyme, fractional excretion of sodium, urine/plasma osmolality, glomerular filtration rate, and the clinical diagnosis of acute renal failure. The urinary output seems to be the most effective parameter to diagnose the condition and act quickly. intraoperative hypovolaemia and hypo-tension seems to be the most important risk factor for renal function[2].

The development of perioperative acute renal failure following trauma or major surgery is a serious complication with a reported incidence of 0.1%–50% and a reported mortality of 17%–84%. Perioperative acute renal failure accounts for one half of all patients requiring acute dialysis.

Ischemic renal tubular damage or acute tubular necrosis may well be the leading cause of acute renal failure in the perioperative period. Ischemic tubular damage is most often caused by an imbalance of oxygen supply and demand to the medullary thick ascending tubular cells. Intrarenal distribution of renal blood flow to the inner medulla can result in decreased oxygen supply, whereas increased tubular reabsorption of solute causes increased oxygen demand. Both increased demand and decreased supply are precipitated during states of inadequate perfusion (prerenal azotemia) such as those resulting from depletion of intravascular volume, maldistribution of systemic flow away from the renal vascular bed, cardiogenic shock, and obstructive vascular conditions[3,4].

Causes of postoperative renal failure includes; cardiogenic shock, sepsis, hypovolemic shock, drug nephrotoxicity and hepatorenal syndome[5].

In this article, we stress on the importance of preserving normal blood pressure and oxygenation during operation which will give constant urine output. Anesthesiologist are not always aware of the reduction in blood pressure and the oxygen saturation in the blood either due to negligence or the lack of monitoring devices such as invasive and non-invasive blood pressure monitoring, pulse oximetry and urine output monitoring specially in major surgeries. It seems that hypo-volemia and hypoxia and unnecessary use of blood transfusion are the major causes of acute postoperative renal failure specially in jaundiced patients.

Our advice to junior anesthesiologists is to preserve the blood volume fluid and keep continuous monitoring of blood oxygen and the urine output. The introduction of a Folly's catheter in major surgery is an important obsession. Once there is low urine output, the anesthesiologist should

work the kidneys by fluid, mannitol and diuretics. Also we encourage the choice of fentanyl and droperidol because that both does not affect the renal blood flow and the systemic blood pressure as do halothane[6].

We used to transfuse blood needlessly in major surgery, here we would like to say to anesthesiologists not to use blood unless it is deliberately needed.

Once anesthesiologists noticed that urine output is unsatisfactory, they should infuse fluid to keep good renal blood flow, use mannitol or furosemide. Most centers use mannitol as the agent of choice for prevention of renal dysfunction when periods of decreased glomerular filtration are anticipated. Pretreatment with intravenous mannitol, continued throughout any ischemic episode, improves subsequent renal blood flow and glomerular filtration[7]. A strong solute diuresis relieves tubular obstruction and reduces tubular cellular swelling[7,8] .

The renovascular and functional effects of loop diuretics are similar to those of mannitol. Furosemide increases renal blood flow and promotes solute excretion[9]. The theoretical benefit of furosemide in the patient with early acute renal failure is to convert oliguric to nonoliguric renal failure. Although this may not improve chances of recovery[10], it avoids some of the complications associated with complete renal shutdown. Furosemide early in renal dysfunction, when only abnormalities in free water clearance are apparent, improves medullary blood flow and free water clearance and identifies patients who are likely to require dialysis by their failure to respond[11-13].

The use of dopamine to enhance renal blood flow and to prevent renal failure deserves consideration. The synergistic effects of dopamine and furosemide (administered after an acute renal injury) have been studied with the conclusion that the combination is more effective than either drug alone[14]. The combination of drugs prompts a brisk diuresis and reduces serum creatinine levels, possibly by enhancing vasodilatation to allow increased delivery of furosemide to the distal tubule[15].

The mainstay to early support is improvement in renal blood flow and maintenance of tubular patency by ensuring adequate cardiac output and blood pressure while promoting renal vasodilatation and glomerular filtration with dopamine, mannitol, and furosemide. If successful, continuous infusion of these agents should be considered.

Special care should be taken in patients having nephrotoxins, sepsis, hepatorenal syndrome, reoperation, impaired cardiac output and if having medications such as contrast medium, cyclosporine, amphotericin, alphaadrenergic agonists and NSADs.

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