

POSTOPERATIVE ACUTE RENAL FAILURE, THE ROLE OF ANESTHESIOLOGISTS.

Salam N Asfar* & Jasim M Salman[#]

*MB,ChB, MSc Professor of Anesthesiology, Department of Surgery, Basrah College of Medicine. [#]MB,ChB, DA, Consultant Anesthesiologist, Al-Sader Teaching Hospital, Basrah, Iraq.

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 hypoperfusion, 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 possibility¹.

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 hypotension seems to be the most important risk factor for renal function².

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 syndrome⁵.

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 halothane⁶.

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 filtration⁷. 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 excretion⁹. 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 recovery¹⁰, 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 respond¹¹⁻¹³.

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 alone¹⁴. 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 tubule¹⁵.

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.

References

- 1-Lote CJ, Harper L, Savage COS: Mechanisms of acute renal failure. *Br. J. Anaesth.* 1996; 77:82-89.
- 2-Bruce K, Roizen Michael F, Ronald A: Association of preoperative risk factors with postoperative acute renal failure. *Anesth Analg* 1994; 78:143-149.
- 3-John BS, Ronald A: Predictive and diagnostic tests of renal failure; a review.. *Anesth Analg* 1994; 78:134-142.
- 4-Cioffi WG, Ashikaga T, Gamelli RL: Probability of surviving postoperative acute renal failure: development of a prognostic index. *Ann Surg* 1984; 200:205-211.
- 5-Tien JC, Kuan YH, Hsuan KJ: prognostic factors of postoperative acute renal failure. *Dialysis & Transplantation*, 1999;29;:11-15.
- 6-Colson P, Capdevilla X, Cuchet D: Does choice of the anesthetic influence renal function during infrarenal aortic surgery? *Anesth Analg* 1992; 74:481-485.
- 7-Frega NS, DiBona DR, Leaf A: Enhancement of recovery from experimental ischemic acute renal failure. *Renal Pathophysiol* 1980. 203.
- 8- Levinsky NG, Bernard DB, Johnston PA: Mannitol and loop diuretics in acute renal failure in Brenner BM, Lazarus JM (eds): *Acute Renal Failure*. Philadelphia, Saunders, 1983, p 712.
- 9- Patak RV, Fadem SZ, Lifschitz MD, Stein JH: Study of factors which modify the development of norepinephrine-induced acute renal failure in the dog. *Kidney Int* 1979; 15: 227.
- 10- Nierenberg DW: Furosemide and ethacrynic acid in acute tubular necrosis. *West J Med* 1980; 133: 163.
- 11-Baek SM, Brown RS, Shoemaker WC: Early prediction of acute renal failure and recovery: II. Renal function response to furosemide. *Ann Surg* 1973; 178: 605.
- 12-Brown RS: Renal dysfunction in surgical patient: Maintenance of the high output state with furosemide. *Crit Care Med* 1979; 7: 63.
- 13- Lameire N, Vanholder R, Biesen WV: Loop diuretics for patients with acute renal failure. *JAMA*, 2002. 288:2599
- 14- Linder A, Cutler RE, Goodman WG: Synergism of dopamine plus furosemide in preventing acute renal failure in the dog. *Kidney Int* 1979; 16: 158.
- 15- Linder A: Synergism of dopamine and furosemide in diuretic-resistant, oliguric acute renal failure. *Nephron* 1983; 33: 121.