Furosemide versus mannitol as a renal protection after adult Cardiac Surgery

Thesis

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أَعُوذُ بِاللَّهِ مِنَ الشَّيْطَانِ الرَّجِيمِ

بِسْمِ اللَّهِ الرَّحْمَٰنِ الرَّحِيمِ

قَالُوا۟ سُبْحَٰنَكَ لَا عِلْمَ لَنَآ

إِلَّا مَا عَلَّمْتَنَآ ۖ إِنَّكَ أَنتَ ٱلْعَلِيمُ ٱلْحَكِيمُ

سُورَةُ البَقَرَة ٣٢

صدق الله العظيم

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Abstract

Background: Occurrence of acute oliguric renal failure in the immediate postoperative period carries important morbidity and mortality after a successful cardiac surgical procedure. Adult cardiac and aortic surgical procedures are especially prone to this complication with the incidence varying between 2% to 15% and the mortality rate as high as 40% to 60%.

Aim of the Work: to compare between furosemide versus mannitol as a renal protection after adult cardiac surgery.

Patients and Methods: This prospective comparative trial was conducted at Academy of Cardiothoracic Surgery Ain Shams University on patients undergoing CABG operation consisting of a total of 50 patients with normal renal function, EF of greater than 40%, normal protein and electrolyte levels.

Results: the study revealed no statistically significant difference between groups according to cystatin-C

Conclusion: The difference between patients given mannitol and patients given furosemide regarding urinary microalbumin, urinary creatinine and serum cystatin-c was insignificant. Addition of mannitol to the priming solution of the cardiopulmonary bypass acts as a renal protector against AKI postoperative. Finally, giving furosemide infusion to patients undergoing cardiac surgery at the beginning of the CPB improves renal perfusion.

Key words: Furoemside, mannitol, renal protection, adult cardiac surgery

Introduction

The kidney is the central organ in regulating body fluid composition, intravascular volume, and excretion of metabolic byproducts. Renal function has been studied extensively in patients undergoing cardiopulmonary bypass (CPB). CPB causes a number of changes in the quantity and distribution of renal blood flow, especially in patients with abnormal renal function. Many physiologic alterations (such as nonpulsatile perfusion, hypothermia, and hemodilution) occur during CPB and may alter renal function. Controversy continues regarding the relative importance of CPB versus predisposing patient factors in causing renal dysfunction after cardiac operations.

The incidence of renal complications in both infant and adult cardiac surgery is reported to be decreasing with improved patient preparation, perfusion techniques, and cardiac performance postoperatively **(Werner et al., 1997).**

Once renal failure is established, however, the mortality rate continues to be over 50% despite supportive care and renal replacement therapy **(Fleming et al., 2001).**

In addition, renal dysfunction significantly lengthens hospitalization, length of stay in critical care units, and total medical cost of cardiac surgical procedures **(Mangano et al., 2003).**

Aim of the Work

The aim of this study is to compare between furosemide versus mannitol as a renal protection after adult cardiac surgery.

Anatomy and Physiology

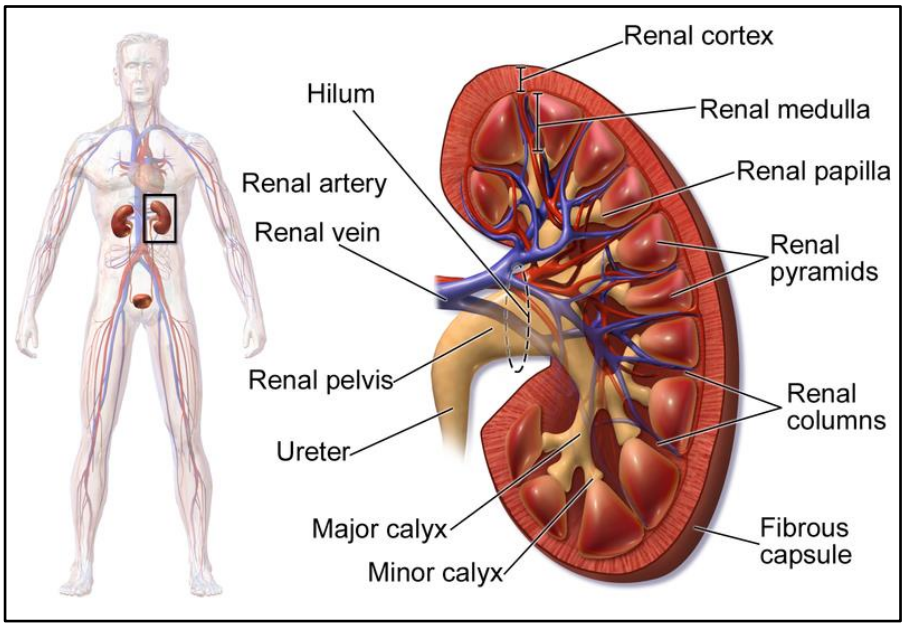

Figure (1): Kidney Anatomy

The basic functional unit of the kidney is the nephron, and each kidney contains over one million of these structures. Anatomically, the kidney is divided into two zones: the cortex, which contains most of the glomeruli, and the medulla, which comprises the collecting system and loops of Henle. The nephron consists of two main structures: a specialized capillary network, the glomerulus, that allows filtration of fluid from plasma devoid of formed cellular components and plasma proteins and a tubular system that collects the filtered fluid and alters its composition to convert the plasma filtrate to urine (**Cotran et al., 2005).**

The kidney and nervous system communicate via the renal plexus, whose fibers course along the renal arteries to reach each kidney.Input from the sympathetic nervous system triggers vasoconstriction in the kidney, thereby reducing renal blood flow. The kidney also receives input from the parasympathetic nervous system, by way of the renal branches of the vagus nerve; the function of this is yet unclear. Sensory input from the kidney travels to the T10-11 levels of the spinal cord and is sensed in the corresponding dermatome.Thus, pain in the flank region may be referred from corresponding kidney (**Bard et al., 2003).**

Blood flowing via the renal artery into the kidney passes through an efferent arteriole to the glomerulus and then exits via an efferent arteriole. Renal blood flow (RBF) accounts for approximately 20% of resting cardiac output. It can be diminished by atherosclerosis of the renal vasculature or increased vasomotor tone due to low cardiac output or in response to administered inotropic agents. The renal vascular bed responds to α-adrenergic stimuli with vasoconstriction that may decrease RBF while maintaining blood pressure. The renal vasculature also contains dopaminergic receptors and is responsive to analogues of atrial natriuretic peptide (ANP), which may specifically alter flow dynamics within the kidney **(Clapp et al., 2009).**

Intraglomerular blood pressure (the difference between the pressures in the efferent and afferent arterioles) drives the filtration of fluid through the capillary endothelium and into Bowman's capsule. The endothelium of the glomerulus has a permeability that is 100 times greater than normal capillaries and is perforated by multiple fenestrations,. These fenestrations prevent the exodus of formed cellular elements from the blood. Glycoproteins guard these exit points electrostatically, repelling negatively charged plasma proteins from the collecting system. The fluid that enters the tubule is an ultrafiltrate similar in composition to plasma but without significant protein content **(Habuka et al., 2014).**

The glomerular filtration rate (GFR) is normally 100 to 200 mL/min in the adult. The GFR is well preserved over a wide range of arterial pressures by autoregulation of glomerular blood pressure. Over 99% of the volume of glomerular filtrate is reabsorbed during its journey from Bowman's capsule, through the loop of Henle, to the collecting ducts at the pelvis of the renal hilum. The reabsorption of water is by passive osmotic diffusion. Certain substances of nutritional value, such as glucose and amino acids, are reabsorbed by active transport and are almost completely conserved by their removal from urine. Proteins finding their way into the glomerular filtrate are often too large to be reabsorbed via conventional transport

mechanisms. Reclamation occurs by pinocytosis by tubular epithelia cells with subsequent breakdown of the protein into amino acids that are returned to the bloodstream. In brief, the nephron filters the plasma and then selectively reabsorbs a significant proportion of the filtrate to produce urine (**Glodny et al., 2009).**

Although GFR is autoregulated across a wide range of arterial blood pressures, urine output is not. Urine output rises in a nearly linear fashion with arterial blood pressure. A rise in arterial blood pressure from 100 to 200 mm Hg will cause a sevenfold increase in urinary output, whereas decreasing blood pressure to 50 mm Hg will cause urine output to nearly cease. The lack of autoregulation of urinary output results in long-term control of blood pressure. As blood pressure rises, urinary output is increased until intravascular volume is depleted and pressure falls to normal levels. The physiologic basis of the relationship between blood pressure and urine output rests both in a slight rise in GFR and more importantly in the rise in peritubular vascular pressures that decreases the reabsorption of filtrate from the tubule (**Guyton 1987).**

In addition to filtering the blood and preventing the loss of important substances from the body by reabsorption, the kidney can significantly alter the excretion of water by