Assessment of Acute Peritoneal Dialysis Efficiency in Pediatric Patients

A thesis

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Marian Aziz Ayoub

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ABSTRACT

In cases of renal failure, renal replacement aims to remove toxins & achieve water, electrolytes & acid-base homeostasis. Acute peritoneal dialysis has many advantages, particularly in children, since it is less demanding an equipment & experience & lack of need for vascular access & anticoagulation.

Removals of fluids, urea & creatinine as well as correction of acidosis and potassium were studied in 21 patients (age from 3months to 14years) undergoing acute peritoneal dialysis for mean duration of 53 hours.

The mean kT/V (urea) of 1.3 & creatinine clearance was 14.6L/1.73m2. ultrafiltration reached 303 ml/kg, with mean of 86 ml/kg . ultrashort (5-10 min) dwells improved ultrafiltration & acidosis correction, with similar clearance of urea & creatinine, Increasing run volumes improved creatinine clearance.

APD is needed & useful in infants & children requiring acute renal replacement.

Key words: Acute peritoneal dialysis – Children – Dwell time – Efficiency – Renal failure – Renal replacement therapy – Ultrafiltration .

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LIST OF ABBREVIATION

APD	=	Acute Peritoneal Dialysis
BSA	=	Body Surface Area
CAPD	=	Continuous Ambulatory Peritoneal Dialysis
CA VH	=	Continuous Arteriovenous Hemofiltration
CAVHD	=	Continuous Arteriovenous Hemofiltration with Dialysis
CCPD	=	Continuous Cycling Peritoneal Dialysis
CVVH	=	Continuous Venovenous Hemofiltration
CVVHD	=	Continuous Venovenous Hemofiltration with Dialysis
ESRD	=	End Stage Renal Disease
HD	=	Haemodialysis
IPD	=	Intermittent Peritoneal Dialysis
IPP	=	Intraperitoneal Pressure
PD	=	Peritoneal Dialysis
PET	=	Peitoneal Equilibration Test
RRT	=	Renal Replacement Therapy

INTRODUCTION AND AIM OF WORK

Renal failure is associated with accumulation of nitrogenous waste products, as well as disturbed fluid, electrolyte & acid –base balance. (Skorecki et al., 2001). Management is rather complex & requires meticulous attention to restoration of fluid , electrolyte & acid-base status as well as removal of uremic toxins (Voget and Avner ,2004).

Renal replacement therapy may be needed for acute or chronic renal failure (Singri et al.,2003)-(parmer .,2003). Particularly in children , peritoneal dialysis has many advantages including technically simplicity , less need for expensive & sophisticated equipment and lack of requirement for vascular access with related difficulties & bleeding risks.

Whereas chronic peritoneal dialysis through a cuffed tunneled peritoneal catheter is often the dialytic mode of choice for children with ESRD (end stage renal disease) , acute peritoneal dialysis will be needed for those with acute or acute on top of chronic renal failure as well as in initial management of those with ESRD starting dialysis in absence of established access (Berket and Fine .,1995).

Several studied have addressed the assessment of chronic peritoneal dialysis (CPD) efficiency & transport characteristics , however , there is a relative lack of data on pediatric acute peritoneal dialysis.

The aim of this work:

To evaluate the efficiency of acute peritoneal dialysis in children regarding :

- Fluid removal.
- Correction of acidosis & electrolyte disturbance.
- Clearance of low molecular weight uremic toxins.

And to determine the effect of using ultrashort (5-10 minutes) dwell time compared to longer dwells.

Acute Renal Failure

Acute renal failure (ARF) represents a rapid decline in renal function sufficient to increase blood levels of nitrogenous wastes and impair fluid and electrolyte balance.

Unlike chronic renal failure (CRF), acute renal failure is potentially reversible if the precipitating factors can be corrected before kidney damage has occurred.

The most common indicator of ARF is azotemia; an accumulation of nitrogenous wastes (urea nitrogen, uric acid, and creatinine) in the blood. In ARF, the GFR is decreased; consequently, excretion of nitrogenous wastes is decreased, and fluid and electrolyte balance cannot be maintained.

ARF is a common threat to seriously ill children in intensive care units, with a mortality rate ranging from 40%-75% ***(Singri et al., 2003)***.Although renal replacement methods are effective in correcting life-threatening fluid and electrolyte disorders, the mortality rate from ARF has not changed substantially since the 1960s ***(Albright, 2001).*** This is probably because ARF is frequently superimposed on other serious situations such as trauma, shock, and sepsis.

Types of Acute Renal Failure

ARF can be caused by a variety of conditions; these include a decrease in blood flow without ischemic injury to the renal tissue, ischemic, toxic, or obstructive tubular injury, and obstruction of urinary tract outflow.

The causes of ARF are categorized as *prerenal, intrinsic,* and *postrenat* ***(Brady et al., 2000; Nally, 2002; Abernethy and Lieberthal, 2003).***

- ***Causes of Acute Renal Failure***

Prerenal

- Hypovolemia Hemorrhage .
- Dehydration .
- Excessive loss of gastrointestinal tract fluids .
- Excessive loss of fluid due to burn injury .
- Decreased vascular filling .
- Anaphylactic shock .
- Septic shock .
- Heart tailure and cardiogenic shock .
- Decreased renal perfusion due to vasoactive mediators, drugs,diagnostic agents .

Intrinsic or intrarenal

- Acute tubular necrosis .
- Prolonged renal ischemia .
- Exposure to nephrotoxic drugs, heavy metals, and organic solvents Intratubular obstruction resulting from hemoglobinuria, myogiobinuria, myeloma light chains, or uric acid casts .
- Acute renal disease (acute glomeruionephritis, pyelonephritis).

Postrenal

- Bilateral ureteral obstruction
- Bladder outlet obstruction

A- Prerenal Failure

Prerenal failure, the most common form of ARF, is characterized by a marked decrease in renal blood flow (RBF). It is reversible once the cause is rapidly identified and promptly corrected before kidney damage occurs.

Causes of prerenal failure include:

1. *profound depletion of vascular volume* (e.g. hemorrhage, severe loss of extracellular fluid volume in severe vomiting, diarrhea, and extensive burns.)
2. *impaired renal perfusion* (e.g. heart tailure, and cardiogenic shock.)
3. *decreased vascular filling* due to increased vascular capacity (e.g. sepsis and anaphylaxis.)
4. intense intrarenal vasoconstriction with *glomerular hypoperfusion* which may occur with some vasoactive mediators (e.g. epinephrine and high doses of dopamine), drugs (e.g. cyclosporine and amphotericin B), diagnostic agents such as those used for cardiac catheterization and intravenous pyelography, and some endotoxins***(Thadhani et al., 1996; Brady et al., 2000; and Nally, 2002).***
5. *impairment of the renal adaptive mechanisms* with conversion of the compensated renal hypoperfusion into a prerenal failure angiotensin-converting enzyme inhibitors (ACEi) reduce the effects of renin on RBF, and when they are used along with diuretics, they may precipitate a prerenal failure in children with an already compromised RBF due to renal vascular disease. As well, nonsteroidal anti-inflammatory drugs (NSAlDs) induce a marked reduction in RBF through the inhibition of prostaglandin synthesis, and hence, can result in prerenal failure in children with diminished renal perfusion ***(Singri, 2003).***

Normally, the kidneys receive 20%-25% Of the cardiac output; This large blood supply is required to remove metabolic wastes and regulate body fluids and electrolytes. The normal kidney can tolerate relatively large reductions in blood flow before renal damage occurs. As RBF is reduced, the GFR decreases and the need tor energy-dependent mechanisms to reabsorb sodium and other substances is consequently reduced; oxygen consumption by the kidney becomes just about that required to keep the renal tubules alive. Any further reduction below 20% of the RBF may result in serious damage in the renal tubules with the high metabolic rate and tubular necrosis may ensue with significant morbidity and mortality (Guyton and Hall, 2000).

B-Intrinsic Renal Failure

Intrinsic or intrarenal failure results from conditions that cause damage to structures within the kidney; glomerular, tubular or interstitial. Acute tubular necrosis (ATN) is the most common form of intrinsic renal failure and implies destruction of tubular epithelial cells with acute suppression of renal function.

The major causes are ischemia associated with prerenal failure ***(Albright, 2001),*** toxic insult to the tubular structures of the nephron (Geriach and Pickworth, 2000), and intratubular obstruction ***(Maddox, 2002).*** Acute glomerulonephritis (AGN) and acute pyelonephritis (APN) are well known causes of intrarenal failure ***(Albright, 2001).***

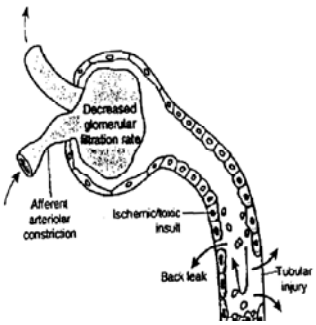

Fig.1 a ;diagrammatic representation of the pathogenesis of ATN

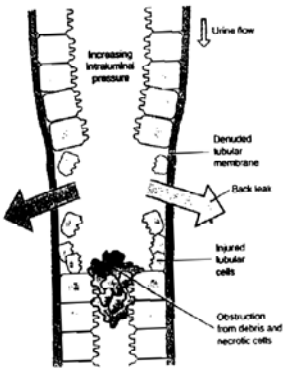

Fig.1 b;diagrammatic representation of the pathogenesis of ATN.

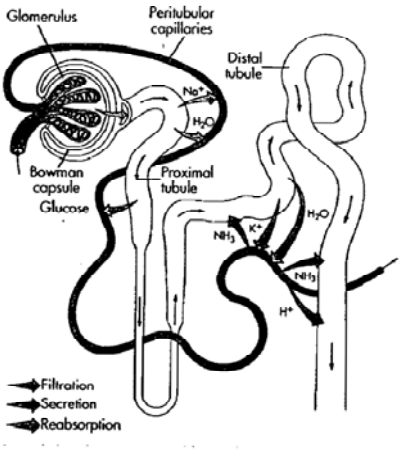

Fig. 2 ;different functional segments of nephron.