

# Outcome of Patients with Acute Oliguria after an Acute Gastroenteritis Episode

TARIQ PERVEZ<sup>1</sup>, SALAHUDDIN QURESHI<sup>2</sup>, NASIB ULLAH SHAH<sup>3</sup>

## ABSTRACT

**Aim:** To know important factors of acute oliguria developing after an episode of acute gastroenteritis.

**Methods:** This comparative study was conducted in the Department of Medicine, Sir Ganga Ram Hospital and Fatima Jinnah Medical College, Lahore from October 2011 to April 2012. A total of 100 patients were included in the study and were divided in two equal groups. Group-A patients were administered low dose dopamine infusion along with fluid replacement and other treatment, whereas group-B patients were given conventional treatment.

**Results:** The postural hypotension was present in group A was 22% and 28% in group B. Serum electrolyte levels were normal in 40(80%) of group A and 44(88%) in group. Hyperkalemia was occurred in 10(20%) in group A and 6(12%) in group B.

**Conclusion:** It is concluded from the study that infectious food was the main cause of gastroenteritis.

**Keywords:** Oliguria, Episode, Gastroenteritis.

## INTRODUCTION

Acute gastroenteritis is endemic in Pakistan<sup>1</sup>. It is one of the major damage to renal failure due to ischemic damage to renal medulla as it often leads to shock. Poor hygienic especially in bazaar cooked food and poor preservation of home cooked food is the major contributing factor. Exposure to animals may also be an important risk factor for acquisition of diarrhoeal diseases especially from organisms such as E-coli<sup>2</sup>.

Early identification of potentially reversible causes of acute oliguria and institution of appropriate therapy are crucial, since the therapeutic window is often small<sup>3</sup>. It may lead to end stage renal failure. End stage renal disease is a devastating physical, economical and social problem for the patients and their family<sup>4</sup>.

The main functional derangement in patients with oliguria is a sudden and severe decrease in the glomerular filtration rate, sufficient to result in increases in the plasma urea and creatinine concentrations, retention of salt and water and the development of acidosis and hyperkalemia<sup>5</sup>. The present study was carried out to know important factors of acute oliguria developing after an episode of acute gastroenteritis

<sup>1</sup>Assistant Professor Medicine, Sir Ganga Ram Hospital/Fatima Jinnah Medical College Lahore

<sup>2</sup>Associate Professor Pharmacology, Sindh Medical College, Jinnah Sindh Medical University, Karachi

<sup>3</sup>Assistant Professor of Medicine Unit-3, Sandeman Provincial Hospital, Bolan Medical College Quetta

Corresponding to Dr. Tariq Pervez, Email. t66p66@hotmail.com; cell: 0300-9639658

## PATIENTS AND METHODS

This randomized study was conducted in the Department of Medicine, Sir Ganga Ram Hospital and Fatima Jinnah Medical College, Lahore from October 2011 to April 2012. A total of 100 patients were included in the study and were divided in two equal groups. Group A patients were administered low dose dopamine infusion along with fluid replacement and other treatment, whereas group B patients were given conventional treatment.

## RESULTS

Out of 100 patients 61(61%) were male and 39(39%) were female patients. The postural hypotension was present in group A was 11(22%) and 14(28%) in group B (Table 1). Serum electrolyte levels were normal in 40(80%) in group A and 44(88%) in group while hyperkalemia was 10(20%) in group A and 6(12%) in group B (Table 2).

Table 1: Postural hypotension in both groups

| Postural hypotension | Dopamine not given | Dopamine given |
|----------------------|--------------------|----------------|
| Absent               | 39(78%)            | 36(72%)        |
| Present              | 11(22%)            | 14(28%)        |

Table 2: Serum electrolyte levels in both groups

| Serum electrolyte | Dopamine not given | Dopamine given |
|-------------------|--------------------|----------------|
| Normal            | 40(80%)            | 44(88%)        |
| Hyperkalemia      | 10(20%)            | 6(12%)         |

## DISCUSSION

Most of the patients developed gastroenteritis after eating bazaar cooked food or reheated stored home food. This is in consistence with many other studies by Centers for Disease Control and Prevention, USA which point to similar which sources of contamination<sup>6-14</sup>.

Leukocytosis was present in 41% of the patients in said study, which may be an indicator of infectious cause of diarrhea in these cases. More patients in the group given dopamine as a part of treatment developed complications (chronic renal failure and hyperkalemia) of acute renal failure. This study failed to show any clear benefit of dopamine as an essential part of treatment of acute renal failure along with other modalities of treatment. Present study may be on a very small scale and may need to be on a larger scale. Many similar studies failed to show any beneficial effect of B-blockers, acetylocholine, arterial natriuretic peptide ordopamine when used in early acute renal failure<sup>15-20</sup>.

## CONCLUSION

It is concluded from the study that infectious food was the main cause of gastroenteritis.

## REFERENCES

1. Ahya SN, Coyne DW. Acute renal failure. In: Ahya SN, Flood K, Paranjothi S editors. The Washington manual of medical therapeutics. 30<sup>th</sup> ed. Philadelphia: Lippincott Williams and Wilkins 2001; 258: 258-63.
2. Warshawsky B, Gutmanis I, Henry B. An outbreak of *E. coli* related to animal contact at a petting zoo. *Can J Infect Dis* 2002; 13: 175-81.
3. Rasneem RA, Kaleem M, Butt MK, Zaheer K, Ashraf HS. Expwience of renal transplantation. *Ann KE Med Coll* 2000; 6(4): 374-5.
4. Uchino S, Kellum JA, Bellomo R. Acute renal failure in critically ill patient. *JAMA* 2005; 294: 13-18.
5. Williams DM, Sreedhar SS, Mickell JJ. Acute kidney failure.. *Arch Pediatr Adolesc Med* 2002;156: 893-900.
6. Tarr PI, Gordon CA, Chandler WL. Shiga-toxin-producing *Escherichia coli* and haemolytic uraemic syndrome. *Lancet* 2005; 365: 1073–86.
7. Hughes AK, Ergonul Z, Stricklett PK, Kohan DE. Molecular basis for high renal cell sensitivity to the cytotoxic effects. *J Am Soc Nephrol* 2002; 13: 2239–2245.
8. Proulx F, Toledano B, Phan V, Clermont MJ, Mariscalco MM, Seidman EG. Circulating granulocyte colony- associated with hemolytic uremic syndrome. *Pediatr Res* 2002; 52: 928–34.
9. Besbas N, Karpman D, Landau D, Loirat C, Proesmans W. A classification of hemolytic uremic syndrome and thrombotic thrombocytopenic purpura and related disorders. *Kidney Int* 2006;; 70: 423–31.
10. Trachtman H, Christen E, Cnaan A. Effect of a toxin-binding agent on diarrhea-associated hemolytic uremic syndrome in children. *JAMA* 2003; 290: 1337–44.
11. Ray P, Acheson D, Chitrakar R, Cnaan A, Gibbs K, Hirschman GH, Christen E, Trachtman H. Basic fibroblast growth factor among children with diarrhea. *J Am Soc Nephrol* 2002; 13: 699–707.
12. Holers VM. The spectrum of complement alternative pathway-mediated diseases. *Immunol Rev* 2008; 223: 300–16.
13. Williams DM, Sreedhar SS, Mickell JJ. . Acute kidney failure.. *Arch Pediatr Adolesc Med* 2002;156: 893-900.
14. Hui-Stickle S, Brewer ED, Goldstein SL. Pediatric ARF epidemiology at a tertiary care center from 1999 to 2001. *Am J Kidney Dis* 2005; 45: 96-101.
15. Agarwal I, Kirubakaran C, Markandeyulu V. Clinical profile and out come of acute renal failure in South Indian children. *J Indian Med Assoc* 2004; 1023: 353–6.
16. Askenazi DJ, Feig DL, Graham NM. 3–5 Year longitudinal follow-up of pediatric patients after acute renal failure. *Kidney Int* 2006; 69: 184-9.
17. Radhakrishnan J, Kiryluk K. Acute renal failures outcomes in children and adults. *Kidney Int* 2006; 69: 17-9.
18. Jayakumar M, Prabahar MR, Fernando EM, . Epidemiologic trend changes in acute renal failure-a tertiary center experience from South India. *Ren Fail* 2006; 28: 405-10.
19. Singh SA, Nalini P, Badhe B. Acute renal failure following snakebite in Children. *J Trop Pediatr* 2003; 49: 308-9.
20. Wanigasuriya KP, Peris-John RJ, Wickremasinghe R. Chronic renal failure in North central province of Sri Lanka. *Trans R Soc Trop Med Hyg* 2007; 101: 1013-7.