

DOI: 10.53555/jptcp.v31i4.5348

TO ANALYSED THE CORRELATION OF ACID- BASE DISTURBANCE ALONG WITH ELECTROLYTE IMBALANCE.

Dr. Pradeep Kumar Kori¹, Dr. Rohit Bhuriya², Dr. Harsha Gupta^{3*}, Dr. Ankush lokhande⁴

 ¹Ex senior resident, Department of General Medicine, N S C B Medical College Jabalpur pradeepkumarkori456@gmail.com
 ²Senior Resident, Department of Emergency Medicine, MGM Medical College Indore rohitbhuriya@gmail.com
 ^{3*}Associate Professor in Anesthesia dept.Chirayu medical college and hospital Bhopal harshagupta13@yahoo.com,9827500788
 ⁴Resident, Department of General Surgery, NSCB, Jabalpur

*Corresponding Author: Dr. Harsha Gupta

*Associate Professor in Anesthesia dept.Chirayu medical college and hospital Bhopal harshagupta13@yahoo.com,9827500788

Abstract-

Introduction-Diarrhea can cause a variety of fluid volume, acid-base and electrolyte abnormality which can cause disruption of various organs functioning and also complicate the disease itself to contribute it to morbidity and mortality.

Aim- To analysed the correlation of acid- base disturbance along with electrolyte imbalance with severity illness toacute kidney injury.

Method- this prospective study done in department of medicine from January 2022 to January 2023 one year period in GMC Shahdol medical college. Total 96 patients included in this study

Results- Among the studied 96 patients, maximum cases belong to the two age groups 20-29 years and 40-49 years with mean age 40.08. Out of studied 96 patients, 50 were males and 46 were females. 88 were having duration of illness <7 days and 8 patients were having duration \geq 7days. 56.3% of study participants were severely dehydrated, 28.1% were moderately dehydrated and only 15.6% were mildly dehydrated.

In our study, serum urea level raised in 31.3% of cases and serum creatinine level raised in only 8.3% of participants in acute diarrheal illness. Acute diarrheal illness patients 77.1% of participants were having below normal Serum sodium level and 58.3% were having below normal serum potassium level in the study. Out of 96 patients, 46.3 % of participants were presented with raised serum urea level with severe dehydration similarly, Serum sodium and potassium was found below the normal value in 92.6% and 72.2% severely dehydrated participants respectively. Out of 96 patients, it was found that 31.3% participants were having metabolic acidosis and only 6.3% were having high anion gap in ABG analysis.Out of 96 patients, 86.7% of them had metabolic acidosis found associated with severe dehydration in acute diarrhoeal condition with p value of < 0.001. In our study, p value of between high anion gap metabolic acidosis and severe dehydration was <0.001and was associated with poor prognosis.

Conclusion- The most common acid-base abnormality apart from normal ABG study, observed in patients with acute diarrheal disease is normal anion gap metabolic acidosis.

Keywords- acute diarrheal illness, arterial blood gas, anion gap

INTRODUCTION

Diarrhea can cause a variety of fluid volume, acid-base and electrolyte abnormality which can cause disruption of various organs functioning and also complicate the disease itself to contribute it to morbidity and mortality.

Maintenance of Acid- base balance requires the cooperation of three major organs: liver, kidneys and lungs. So, it can be said that the disease of these organs can contribute to an important proportion of acid- base disturbances.

The present study aims to evaluate various acid- base disturbancein its homeostasis in the patients of acute diarrheal illness.

Also, the study tries to establish a prognostic role of above- mentioned disturbance for early correction of acid-base changes and electrolyte abnormalities and decreasing the severity of illness.

Metabolic acidosis is the most characteristic acid- base disorder known to occurs in patient with diarrhea. The pathophysiology says, that the loss of bicarbonate stores through diarrhea or renal vascular wasting leads to a metabolic acidosis state characterized by increased plasma chloride concentration and decreased plasma bicarbonate concentration Primary metabolic acidosis that occurs as a result of marked increase in endogenous acids when excretion is impaired by renal insufficiency are characterized by decreased plasma bicarbonate concentration and increased anion gap without hyperchloremia.

AIMS AND OBJECTIVES

To analysed the correlation of acid- base disturbance along with electrolyte imbalance with severity illness toacute kidney injury.

OBSERVATIONS AND RESULTS

-In present study 20 year to more than 80-year participants were enrolled and all age group had almost similar around 15% of participation. Gender wise almost equal participation of male and female having history of acute diarrheal disease.

Table 1. Investigation					
Parameter				Frequency	Percent (%)
	<20	mg%	(below	5	5.2
	normal)				
	20-45 mg% (normal)			61	63.5
Serum Urea	>45mg%		(above	30	31.3
	normal)			I	I
Serum Creatinine	< 0.70			38	39.6
	0.70-1.30			50	52.1
	>1.30			8	8.3
Serum Sodium	<136			74	77.1
	136-145			21	21.9
	>145			1	1.0
Serum Potassium	<3.6	mg%	(below	56	58.3
	normal)	-			
	3.6-5.0 mg% (normal)			40	41.7
	<09mg%		(below	14	14.6
	normal)				
Serum Calcium	9-11 mg%	(normal)		68	70.8
	>11mg%		(above	14	14.6
	normal)				
Total				96	100

Table 1 : Investigation

-In Acute Diarrheal illness, serum urea level raised in 31.3% of cases and serum creatinine level raised in only 8.3% of participants. 77.1% of participants were having below normal Serum sodium

level and 58.3% were having below normal serum potassium level in the study. 14% of study participants were having below normal calcium level.

-56.3% of study participants were severely dehydrated, 28.1% weremoderately dehydrated and only 15.6% were mildly dehydrated.



-Almost half (49%) of the participants were showing sinus tachycardia, 4.2% with sinus tachycardia with U wave and 5.2% presented with sinus tachycardia with flat T wave rest other (41.7%) were showing normal ECG finding.

-In ABG analysis 31.3% participants were having metabolicacidosis and only 6.3% were having high anion gap.

-Age was not found statistically associated with dehydration inacute diarrheal conditions. No specific age wise trend was observed.

-Gender was not found statistically associated with dehydration in acute diarrheal conditions. Almost similar proportion of male and female was presented in all dehydration category.

-Severe dehydration (14.8%) was found significantly higher inpatients with \geq 7 days of acute diarrheal disease.

-Serum Urea, Serum sodium and potassium was found statistically associated with severity of dehydration and Serum creatinine and serum calcium had shown no statistical association. 46.3 % of participants were presented with raised serum urea level with severe dehydration similarly, Serum sodium and potassium was found below the normal value in 92.6% and 72.2% severely dehydrated participants respectively.

-Sinus tachycardia, sinus tachycardia with U wave and with flat T wave was found statistically associated. Majority of ECG changes were found in severely dehydrated patients.

 Table 11: Association between categories of dehydration and ABGReadings

Metabolic acidosis (86.7%) was found statistically associated with severe dehydration in acute diarrhoeal condition. Similarly, very high chances of high anion gap was reported in severe dehydration.

-total mortality due to acute diarrheal illness is 1.04%.

DISCUSSION

An observational study was conducted on 96 patients admitted in general ward at Department of Medicine, GMC Shahdol. These patients were assessed at the time of admission by a detailed history taking and duration of illness also giving due importance to comorbidities like diabetes, chronic renal disease, COPD, any drug history, etc. A thorough clinical examination was done for patients which included general examination, vitals, and other systemic examinations. Patients were also categorized according to hydration status into mild, moderate and severe using system adopted from Mandell, Douglas Text Book on Principles and Practice of Infectious Diseases Out of 96 patients of acute diarrheal illness, 50 were females and 46 were males. The mean duration of diarrhea at presentation was less than 3 days.

Among the 96 patients studied 4 patients (2 Men &2 Women) had renal failure (serum creatinine more than 2mg%) at initial presentation to the hospital (25). The mean duration of diarrhea on admission, in this population was 3 days. None of the patients developed renal failure after admission to hospital.

The ABG values of all 96 patients were interpreted in a systematic way as previously described.

Out of the 96 patients studied, 66 patients had a normal ABG values, 24 patients had a normal anion gap metabolic acidosis, 6 patients had increased anion gap metabolic acidosis.

Hence it is shown that, though normal ABG study was found to be prevalent in maximum number of patients but NORMAL ANION GAP METABOLIC ACIDOSIS IS THE MOST COMMON ACID-BASE

ABNORMALITY in acute diarrheal illness.

The reason for a normal anion gap metabolic acidosis, as described previously is loss of bicarbonate in diarrheic stools.

The other findings noted in this population of patients included

- i. Hyperchloremia findings (Sr. Chloride > 105 mEq/L) Rudman etal.
- ii. Normal Na+ and K+ values in maximum patients.
- iii. A less severe acidosis (i.e., pH > 7.20) in most (22 out of 24) patients
- iv. Expected range of respiratory compensation.

Hyperchloremia occurring in this population was a compensatory response to loss of bicarbonate in stools, so as to maintain the electro neutrality of Extra Cellular Fluid (ECF) (Hence referred to as HYPERCHLOREMIC ACIDOSIS) **Zalunardo et al, Wang F Butler et al.**, ^[20]. Even though serum sodium and potassium levels were normal, hypokalemia can also be anticipated, because patients with acute diarrheal disease lose potassium through GIT. Likewise, abnormalities in serum sodium levels can also be anticipated.

22 out 24 patients had a pH above 7.20 and all had respiratory compensation in the expected range. The next common acid-base-disturbance observed was an increased anion gap metabolic acidosis (6 patients).

A patient with acute diarrheal illness can develop increased anion gap metabolic acidosis for the following reasons.

- i. Development of renal failure with retention of acidic anions like sulphate, phosphate etc.
- ii. Lactic acidosis occurring as a result of tissue hypoperfusion.
- iii. Keto acidosis due to starvation.

All patients in this group were evaluated with the above possibilities in mind.

It was observed that all 4 patients had renal failure (Sr. creatinine >2.0 mg%) and their urine tested negative for ketones. Serum albumin wasmeasured in this population of patients (because albumin

is a normalanionic constituent of plasma and perturbations in albumin level mayalter anion gap) and found to be with in the normal reference range. Serum lactate could not be measured. Hence one explanation that couldbe offered for the increased anion gap metabolic acidosis in this settingwas renal failure. Serum chloride level was found to be normal inmaximum patients (an expected finding in increased anion gap acidosis).Na+ and K+ levels were found to be normal.

During this study, maximum number of patients exhibited wasnormal ABG study in acute diarrheal illness (66 patients).

The following 3 possibilities must be considered when one encounters a normal ABG analysis in acute diarrhea :

- i. A patient might have a mild diarrheal illness, so that there is only a minimal bicarbonate loss, which is of no biochemical significance.
- ii. A combination of metabolic acidosis (due to bicarbonate loss in stools) and metabolic alkalosis (due to loss of acid in vomits) may occur in patients with acute diarrheal diseases.

So, a normal ABG study in the clinical context of severe vomiting and diarrhea should suggest a combination of metabolic acidosis and metabolic alkalosis. In this clinical situation pH, pCO_2 , HCO_3^- , AG all will be normal.

iii. A combination of high anion gap acidosis (ex. renal failure, lactic acidosis) and metabolic alkalosis (due to vomiting) may coexist. Here pH, pO2, pCO₂ and HCO₃⁻ will be normal, but anion gap will be high.

All 66 patients who had normal ABG study were clinically suffering from a milder to moderate degree of diarrhea and dehydration and vomiting was not a prominent manifestation. So, a milder diarrheal illness may be postulated as the reason behind the normal ABG study, than a mixed acid base disorder. No significant difference in the clinical presentation of each of the3 groups of patients could be noted.

This study also aims to analyse the acid-base changes that occur inpatients developing renal failure due to acute diarrhea also which was also analysed by a study conducted by **Shivkumar et al**. Among the 96 patients studied 4 patients had renal failure at presentation to hospital. All 4 patients demonstrated a HIGH ANION GAP METABOLIC ACIDOSIS. 3 out of 4 patients had severe metabolic acidosis (pH < 7.2), **Thomas et al**.

In all 4 patients urine tested negative for acetone and 48 serum albumin was normal.

It was found that 9 out of 96 patients with acute diarrhea had a severe metabolic acidosis i.e., pH < 7.2 in ABG study, similar resultswere also conducted by **Mara Nitu et al**. Hence the incidence of severe metabolic acidosis in patients with acute diarrhea was 8.64%. Among these 9 patients; 4 patients had renal failure and 5 patients had normal renal function. Hence severe metabolic acidosis occurred in both groups of patients with acute diarrhea (i.e., patients with renal failure and patients with normal renal function), also shown by a study conducted by **V.K.Praveen Kumar et al**.

CONCLUSION

The most common acid-base abnormality apart from normal ABG study, observed in patients with acute diarrheal disease is normal anion gap metabolic acidosis.

Other acid-base patterns observed include increased anion gap metabolic acidosis. A normal ABG must be interpreted in the clinical context because mixed acid base disorders may produce normal values in ABG analysis. Increased anion gap metabolic acidosis is the acid-base abnormality observed in post diarrheal ARF. In acute diarrheal illness and post diarrheal acute renal failure, metabolic acidosis is a prognostic factor and its outcome can be improved with early recognition and correction.

REFERENCES

- 1. Acute Diarrheal Disease, Epidemiology of Communicable Diseases 25th edition, ``PARKS TEXT BOOK OF PREVENTIVE AND SOCIAL MEDICINE``.
- 2. Diarrhea B.S. Ramakrishna, Gastroenterology, 12th Eiditon, ``API TEXT BOOK OF MEDICINE``.
- 3. Hydrogen Ion Concentration ``INTERPRETATION OF BLOOD GASES`` ``ROBERT L.WILKINS, 7th Edition, Clinical Assessment in Respiratory Care`` by Wilkins, Krider and Sheldon.
- 4. Introduction to Acid Base Physiology 7. ``Understanding Acid Base Balance`` by Benjamin Abelow M.D.,
- 5. Transport of oxygen and Carbondioxide in Blood and Body Fluids– 40. `TEXT BOOK OF MEDICAL PHYSIOLOGY`` 14thEdition, Guyton and Hall.
- 6. ``Understanding Acid Base Balance`` by Benjamin Abelow M.D.,
- 7. Acidosis and Alkalosis–42, Thomas B. Dubose JR. ``HARRISONS PRINCIPLES OF INTERNAL MEDICINE`` Volume 1, 20th Edition.
- 8. Buffering of Hydrogen Ions in Body Fluids, Regulation of Acid Base Balance 30, `TEXT BOOK OF MEDICAL PHYSIOLOGY`` 14th Edition, Guyton and Hall.
- Role of the Kindeys in Acid Base Balance, Urinary Stones, Nephrocalcinosis and Renal Tubular Acidosis – 20.13 Robert J. Unwin William G. Robertson. ``OXFORD TEXT BOOK OF MEDICINE`` 5th Edition.
- 10. Acute Renal Failure due to Acute Diarrheal Diseases –S. Shivakumar, M.A. Muthusethupathi, JAPI Vol.38. Feb-1990.
- 11. Gastrointestinal causes of Metabolic Acidosis, Chappter 16, ``UNDERSTANDING ACID BASE BALANCE`` by BenjaminAbelow M.D.,
- 12. Clinical Acid-Base Disorders 2.6. by Biff. F.Palaer, Robert G. Narins and Jerry Yee in ``OXFORD TEXT BOOK OF CLINICAL NEPHROLOGY`` 5th Edition, Volume-1.
- 13. Metabolic causes of Metabolic Acidosis by Benjamin Abelow in ``UNDERSTANDING ACID BASE BALANCE`` by BenjaminAbelow.
- 14. Metabolism of Ketone Bodies, in Chapter 13, Lipids Part II, ``TEXT BOOK OF BIOCHEMISTRY`` Vasudevan, 10th Edition.
- 15. Clinical Acid-base Disorders 2.6. by Biff. F.Palaer, Robert G. Narins and Jerry Yee in ``OXFORD TEXT BOOK OF CLINICAL NEPHROLOGY`` 5th Edition, Volume-1.
- 16. Disorders of Hydrogen Ion Homeeostasis In ``CLINICAL CHEMISTRY`` 5th Edition by William J. Marshall and Stephen K. Bengert.
- 17. Interpretation of Arterial Blood Gases, Chapter 6, by Robert L. Wilkins in ``CLINICAL ASSESSMENT IN RESPIRATORY CARE`` 4th Edition, Wilkins, Krider and Sheldon.
- 18. Acid-Base Disturbances in ``THE WASHINGTON MANUAL OF MEDICAL THERAPEUTICS`` 36th Edition.
- 19. Limitations of Compensation for Acid Base Disorders in Interpretation of Arterial Blood Gases, Chapter 6, by Robert L.WILKINS,8th Edition, ``CLINICAL ASSESSMENT IN RESPIRATIORY CARE`` by Wilkins, Krider and Sheldon.
- 20. ``Anion Gap`` in Acidosis and Akalosis by Thomas D. Dubose, JR. in 20th Edition HARRISON'S PRINCIPLES OF INTERNAL MEDICINE Volume 1, 20th Edition.
- 21. Metabolic Acidosis in Acid-base disturbances in ``THE WASHINGTON MANUAL OF MEDICAL THERAPEUTICS``31st Edition.
- 22. Treatment of Metabolic Acidosis. Thomas D. Dubose, JR. in 19th Edition, HARRISON'S PRINCIPLES OF INTERNAL MEDICINE Volume 1, 19th Edition.
- 23. Base Excess and Base Defecit, ``CLINICAL ASSESSMENT IN RESPIRATORY CARE`` 4th Edition, Wilkins, Krider and Sheldon.
- 24. Arterial Blood Sampling ``CLINICAL ASSESSMENT IN RESPIRATORY CARE`` 4th Edition, Wilkins, Krider and Sheldon.
- 25. Acute Renal Failure due to Acute Diarrheal Diseases -S. Shivakumar, M.A. Muthusethupathi,

JAPI Vol.38. Feb-1990.

- 26. Metabolic Acidosis in Renal Failure, Acidosis and Akalosis by Thomas D. Dubose, JR. in 20th Edition HARRISON'S PRINCIPLES OF INTERNAL MEDICINE Volume 1, 20thEdition.
- 27. ``ACID BASE CHEMISTRY AND BUFFERING`` by Madias NE.
- 28. Halpern ML. ``BIOCHEMISTRY AND PHYSIOLOGY OF AMMONIUM EXCRETION``. Chapter 76 in ``THE KIDNEY`` by knepper MA.
- 29. Winter SD. The fall of serum anion gap. Arch. Internal Medicine 1990; 150:311-313.
- 30. ``Mixed-Acid Base Disorders, Clinical Examples`` Harrington JT. Allen S. J., Okoko B., Martinez E., Gregorio G., Dans L. F. Probiotics for Treating Infectious Diarrhea. Cochrane DatabaseSystematic Reviews. 2004;(2):CD003048. [PubMed]
- 31. Peter G., Myers M. G. the National Vaccine Advisory Committee, and the National Vaccine Program Office. . Intussusception, Rotavirus, and Oral Vaccines: Summary of a Workshop. Pediatrics. 2002;110:e67. [PubMed]
- 32. Ronsmans C., Bennish M. L., Wierzba T. Diagnosis and Management of Dysentery by Community Health Workers. Lancet. 1988;8610:552–55. [PubMed]
- 33. Ruxin J. N. Magic Bullet: The History of Oral RehydrationTherapy. Medical History. 1994;38:363–97. [PMC free article] [PubMed]
- 34. Ryan E. T., Calderwood S. B. Cholera Vaccines. Clinical Infectious Diseases. 2000;31:561–65. [PubMed]
- 35. Salam M. A. Antimicrobial Therapy for Shigellosis: Issues on Antimicrobial Resistance. Japanese Journal of Medical Science andBiology. 1998;51(Suppl.):S43–62. [PubMed]
- 36. Santos ., Victora C. G., Martines J., Goncalves H., Gigante D. P., Valle N. J., Pelto G. Nutrition Counseling Increases Weight Gain among Brazilian Children. Journal of Nutrition. 2001;131:2866–73. [PubMed]
- 37. Snyder J. D., Merson M. H. The Magnitude of the Global Problem of Acute Diarrhoeal Disease: A Review of Active Surveillance Data. Bulletin of the World Health Organization. 1982;60:604–13. [PMC free article] [PubMed]
- 38. Tucker A. W., Haddix A. C., Bresee J. S., Holman R. C., Parashar U. D., Glass R. I. Cost-Effectiveness Analysis of a Rotavirus Immunization Program for the United States. Journal of the American Medical Association. 1998;279:1371–76. [PubMed]
- 39. Victora C. G., Bryce J., Fontaine O., Monasch R. Reducing Deaths from Diarrhoea through Oral Rehydration Therapy. Bulletin of the World Health Organization. 2000;78:1246–55. [PMC free article][PubMed]
- 40. WHO (World Health Organization). 1997. *Health and Environment in Sustainable Development Five Years after the Health Summit.* WHO/EHG/97.8. Geneva: WHO.
- 41. ——. 2004. Joint Statement: Clinical Management of Acute Diarrhoea. WHO/FCH/CAH/04.7. Geneva: WHO; New York: UNICEF.
- 42. Bandis K. Acid base physiology. Accessed March 2011 at: http:// www.anaesthesamcq.com/ Acid basebook/ABindex.php.
- 43. Kraut JA, Madias NE. Approach to patients with acid-base disorders. Respiratory care . 2001: 46:392-403.
- 44. Hamm LL, DuBose TD. Disorders of acid-base balance. In: Yu ASL, Chertow GM, Luyckx VA, Marsden PA, Skorecki K, Taal MW, eds. *Brenner and Rector's The Kidney*. 11th ed. Philadelphia, PA: Elsevier; 2020: chap 16.
- 45. Palmer BF. Metabolic acidosis. In: Feehally J, Floege J, Tonelli M, Johnson RJ, eds. *Comprehensive Clinical Nephrology*. 6th ed. Philadelphia, PA: Elsevier; 2019: chap 12.
- 46. Seifter JL. Acid-base disorders. In: Goldman L, Schafer AI, eds. *Goldman-Cecil Medicine*. 26th ed. Philadelphia, PA: Elsevier; 2020: chap 110.

RELATED STUDIES

47. Metabolic acidosis in acute renal failure following A.D.D., - An important prognostic factor? M.A. Muthusethupathi, S. Shivakumar et al., JAPI Vol. 40 1992.

- 48. Acute Renal Failure due to Acute Diarrheal Diseases. M.A. Murthusethupathi, S. Shivakumar et al., JAPI Vol 38. Feb 1990.
- 49. Clinical studies in Asiatic cholera, Preliminary observations Carpenter CCJ, Mitra PP, Such R. Nov. 1962 March 1963, Bull Johns Hopkins hosp.
- 50. Clinical Studies in Asiatic Cholera II, Development of 2:1 Saline Lactate Regimen. Chrpenter CCJ, et al., Bull Johns Hopkins Hosp. 1966.