Journal of Advanced Medical and Dental Sciences Research

@Society of Scientific Research and Studies

Journal home page: www.jamdsr.com doi: 10.21276/jamdsr ICV 2018= 82.06

(e) ISSN Online: 2321-9599; (p) ISSN Print: 2348-6805

Original Research

Assessment of serum electrolytes in patients with inflammatory bowel disease

Himanshu Tyagi

Msc, Phd, Assistant Professor, Department of Biochemistry, K M Medical College, Mathura, U.P., India

***ABSTRACT*:**

Background: Inflammatory bowel disease (IBD) represents a chronic inflammatory disorder of the intestine. The present study was conducted to assess serum electrolytes in patients with inflammatory bowel disease. **Materials & Methods:** The present study was conducted in the department of Biochemistry. It comprised of 48 patients of IBD of both genders. 5 ml of blood was taken from all subjects for estimation of serum electrolytes in using ion selective electrolyte technique. **Results:** The mean serum sodium level in group I was 131.4 mEq/l and in group II was 138.1 mEq/l, serum potassium level was 3.94 mEq/l and in group II was 4.18 mEq/l, serum chloride level was 104.5 mEq/l and in group II was 101.2 mEq/l**.** The difference was significant ($p< 0.05$). **Conclusion:** Authors found significant decrease in serum sodium potassium and increase in serum chloride level in patients of IBD.

Key words: Sodium, Potassium, chloride.

Received: 20-07- 2019 Accepted: 23-08-2019

Corresponding author: Himanshu Tyagi, Msc, Phd, Assistant Professor, Department of Biochemistry, K M Medical College, Mathura, U.P., India

This article may be cited as: Tyagi H. Assessment of serum electrolytes in patients with inflammatory bowel disease. J Adv Med Dent Scie Res 2019;7(9):248-250.

INTRODUCTION

Inflammatory bowel disease (IBD) represents a chronic inflammatory disorder of the intestine, generally classified by histopathological and clinical features into two major entities: Crohn's disease (CD) and ulcerative colitis (UC). UC is characterized by diffuse mucosal inflammation limited to the colon. It involves the rectum in about 95% of cases and may extend proximally in a symmetrical, circumferential, and uninterrupted pattern to involve parts or the whole of the large intestine.[1] On the other hand, CD is characterized by asymmetric, transmural and occasionally granulomatous inflammation affecting the gastrointestinal (GI) tract, most commonly the terminal ileum and colon, with the potential for systemic and extraintestinal complications. CD-associated transmural inflammation often leads to fibrosis, obstructive complications, sinus tracts and fistulae, not typically seen in UC.[2]

Despite the substantial cost of IBD to both patients and society, curative, medical interventions have yet to be discovered. The development and persistence of IBS symptoms have been acknowledged as multifactorial in nature, making treatment of the disorder a complicated, clinical endeavor. Approaches are based on the reduction of patient symptomatology, and current pharmacological management often provides suboptimal relief.[3] IBD is recognized as a multifactorial disorder, with the following among the proposed mechanisms contributing to symptomatology: gastrointestinal dysmotility, inflammation, visceral hypersensitivity, and altered intestinal microbiota. Diet and stress exposure (including early life events) have been proposed as contributing factors to this heterogeneous disorder.[4]

The present study was conducted to assess serum electrolytes in patients with inflammatory bowel disease.

MATERIALS & METHODS

The present study was conducted in the department of Biochemistry. It comprised of 48 patients of IBD of both genders. Equal number of control was also taken. The study was approved from institutional ethical committee. All subjects were informed regarding the study and written consent was obtained.

Data such as name, age, gender etc. was recorded. 5 ml of blood was taken from all subjects for estimation of serum electrolytes in using ion selective electrolyte technique. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of subjects

Total- 96		
Groups	**Group I (Cases)**	**Group II (Control)**
Number	48	48

Table I shows that both groups had 48 subjects each. Group I had cases and group II had controls.

Table II Estimation of serum electrolytes

Serum electrolytes (mEq/l)	Group I (Cases)	Group II (Control)	P value
Sodium	131.4	138.1	0.05
Potassium	3.94	4.18	0.02
Chloride	104.5	101.2	0.12

Table II, graph I shows that mean serum sodium level in group I was 131.4 mEq/l and in group II was 138.1 mEq/l, serum potassium level was 3.94 mEq/l and in group II was 4.18 mEq/l, serum chloride level was 104.5 mEq/l and in group II was 101.2 mEq/l**.** The difference was significant ($p< 0.05$).

DISCUSSION

INFLAMMATORY BOWEL DISEASE (IBD) Is A Chronic Inflammatory Intestinal Disorder Encompassing Two Major Entities: Crohn's disease and ulcerative colitis. Intestinal inflammatory processes reduce the absorption of sodium, chloride and calcium, while they increase potassium secretion. In addition, mild to severe metabolic alkalosis may occur in IBD patients, mainly depending on the severity of the disease and the part of the gastrointestinal tract being affected.

IBD-associated mucosal inflammation and the consequent impaired secretion and absorption of electrolytes often result in electrolytic and acid-base imbalance in IBD patients. The main transport abnormality is the decrease in net sodium and chloride absorption, resulting in impaired water absorption or secretion. Diet and stress exposure (including early life events) have been proposed as contributing factors to this heterogeneous disorder.[5] Because stress has been identified as a mechanism in the development of IBS, the major components of the stress response system, the autonomic nervous system (ANS) and the hypothalamic-pituitary-adrenal (HPA) axis, have been the subject of numerous investigations of IBD. Lastly, genetic predisposition and environmental interactions, such as familial susceptibility and psychosocial stressors, have been implicated in the multifactorial pathogenesis of IBD.[6] The present study was conducted to assess serum electrolytes in patients with inflammatory bowel disease.

In present study, both groups had 48 subjects each. Group I had cases and group II had controls. The key determinant of colonic water absorption is the rate of Na^+ absorption. This can be either electrogenic via the epithelial sodium channel (ENaC) or electroneutral via parallel Na^+/H^+ and Cl^-/HCO_3^- exchange.[7] Specifically, in electrogenic absorption of Na^+, apical Na^+ entry is passive, channel-mediated and inhibited by amiloride, while basolateral Na^+ extrusion is mediated by Na^+-K^+-ATPase (the electrogenic 'Na^+ pump'). On the other hand, in electroneutral NaCl absorption, apical Na^+ uptake is mediated by Na^+-H^+ exchange, most likely linked with intracellular pH to apical Cl^-/HCO_3^- exchange. As for the place in which these absorptive processes are located, the electrogenic absorption is confined to the surface epithelium and upper crypts of the distal colon, while the electroneutral one takes place in both crypts and surface epithelium of the proximal and distal colon.[8]

We found that mean serum sodium level in group I was 131.4 mEq/l and in group II was 138.1 mEq/l, serum potassium level was 3.94 mEq/l and in group II was 4.18 mEq/l, serum chloride level was 104.5 mEq/l and in group II was 101.2 mEq/l**.** Lucas *et al*[9] studied the net electrolyte and water transport in the rectum and the rectal PD in 3 groups before and 5 h after a simple i.v dose of steroids. The first group consisted of 9 patients with active UC, the second of 6 patients with inactive UC and the third of 17 control subjects. A strong reduction in PD and net sodium absorption was noticed in patients with active UC, while in those with inactive UC these transport parameters were normal. Similarly, bilateral sodium isotope flux studies in distal colonic mucosa demonstrated decreased net sodium absorption in untreated UC patients due to a reduced mucosa to serosa unidirectional flux. *In vitro* measurements of the net transport and simultaneous bidirectional flux rates of water and electrolytes across the human colonic epithelium demonstrated that in UC the colon becomes less absorptive and more secretory. Specifically, in the active phase of UC colon absorbs less water and sodium and secretes more potassium.[10]

CONCLUSION

Authors found significant decrease in serum sodium potassium and increase in serum chloride level in

patients of IBD. In contrast to other secretory diarrheas, IBD has been associated with mild or severe metabolic alkalosis depending on the severity of the inflammation and the part of the GI tract being affected. The alkalosis is attributed to the decreased bicarbonate secretion of the colon when it is affected. Hence, active UC may increase patient pH in comparison with CD that might not affect the colon. Large-scale epidemiological studies are required for a better insight into electrolyte and acid-base disorders in IBD patients.

REFERENCES

1. Podolsky DK. Inflammatory bowel disease 1999: present and future promises. Curr Opin Gastroenterol. 1999;15:283–284.
2. Kornbluth A, Sachar DB. Ulcerative colitis practice guidelines in adults. American College of Gastroenterology, Practice Parameters Committee. Am J Gastroenterol. 1997;92:204–211
3. Debongnie JC, Phillips SF. Capacity of the human colon to absorb fluid. Gastroenterology 1978;74:698-703.
4. Kockerling A, Sorgenfrei D, Fromm M. Electrogenic Na+ absorption of rat distal colon is confined to surface epithelium: a voltagescanning study. Am J Physiol 1993;264(5 Pt 1):C1285-C1293.
5. Barbry P, Hofman P. Molecular biology of Na+ absorption. Am J Physiol 1997;273(3 Pt 1): G571-G585.
6. Halm DR, Halm ST. Secretagogue response of goblet cells and columnar cells in human colonic crypts. Am J Physiol Cell Physiol 2000;278:C212-C233.
7. Sandle GI. Salt and water absorption in the human colon: a modern appraisal. Gut 1998;43:294-299. 10. Kockerling A, Fromm M. Origin of cAMP-dependent Cl- secretion from both crypts and surface epithelia of rat intestine. Am J Physiol 1993;264(5 Pt 1):C1294-C1301. 8. Dawson DC. Ion channels and colonic salt transport. Annu Rev Physiol 1991;53:321-339.
8. Lucas ML, Cooper BT, Lei FH, et al. Acid microclimate in coeliac and Crohn's disease: a model for folate malabsorption. Gut 1978;19:735-742.
9. Press AG, Hauptmann IA, Hauptmann L, et al. Gastrointestinal pH profiles in patients with inflammatory bowel disease. Aliment Pharmacol Ther 1998;12:673-678.
10. Rampton DS, Sladen GE, Youlten LJ. Rectal mucosal prostaglandin E2 release and its relation to disease activity, electrical potential difference, and treatment in ulcerative colitis. Gut. 1980;21:591–596.