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Study of Electrolytes and Blood Gas changes in Acute and Acute-on-Chronic Intestinal Obstruction

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ABSTRACT

Introduction: *Intestinal obstruction is the significant mechanical impairment or complete arrest of the passage of contents through the intestine. It accounts for 20% of all acute surgical admissions. The study was conducted to find out the electrolytes pattern and arterial blood gas changes among different variety of intestinal obstruction.* ***Methods:*** *This cross-sectional observational study included 200 subjects in acute and acute-on-chronic intestinal obstruction in different general surgical wards in Dhaka Medical College Hospital, Dhaka during July to December, 2013.* ***Results:*** *The highest number of the patients with acute intestinal obstruction presented belongs to 31-40 years age group (35, 32%). Majority (64, 32%) of causes of intestinal obstruction were adhesion followed by intestinal TB (57, 28.5%). In acute intestinal obstruction, 76 (69.0%) patients had hyponatraemia and 80 (72.7%) had hypokalaemia during admission, while most of hyponatraemia 92 (83.6%) and hypokalaemia 89 (80.9%) were corrected after resuscitation. Majority of the subjects had alkalosis (70.9%) and decreased $PaCO_2$ (70.9%) before resuscitation. After resuscitation, patients with 80.9% alkalosis and 78.0% $PaCO_2$ returned to normal. In acute-on-chronic intestinal obstruction, 71 (78.9%) had hyponatraemia and 74 (82.2%) had hypokalaemia before resuscitation, while majority of hyponatraemia (70, 77.8%) and hypokalaemia (68, 75.5%) were corrected following resuscitation. Majority of the subjects had alkalosis (67, 74.4%) and decreased $PaCO_2$ (66, 73.3%) before resuscitation. Following resuscitation P^H, $PaCO_2$ of patients returned to normal by 75.5% and 73.3% respectively.* ***Conclusion:*** *Acute and acute-on-chronic obstruction patients had hyponatraemia and hypokalaemia with abnormalities in Arterial Blood Gas (ABG) on admission. Correction of electrolytes imbalance before surgery reduces mortality and morbidity.*

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INTRODUCTION

Acute intestinal obstruction occurs when there is an interruption in the forward flow of intestinal contents. This interruption can occur at any point along the length of the gastrointestinal tract, and clinical symptoms often vary based on the level of obstruction. Acute-on-chronic intestinal obstruc-tion may present with short history of abdominal distension against background of pain and constipation. It usually spread from large gut to involve small bowel. The condition is often treated conservatively over a period of 2–5 days with the patient's progress regularly monitored by an assigned physician. Surgical procedures are performed in life-threatening cases.[1,2] The clinical presentation generally includes nausea and emesis, colicky abdominal pain and a failure to pass flatus or bowel movements. The classic physical examination findings of abdominal distension, tympani to percussion, and high-pitched bowel sounds suggest the diagnosis. Radiologic imaging can confirm the diagnosis and can also serve as useful adjunctive investigations when the diagnosis is less certain.[3] Combined water and electrolyte depletion may occur from gastrointestinal losses due to vomiting and sequestration of large volume of fluid in intestine. Proximal to the point of obstruction, the intestinal tract dilates as it fills with intestinal secretions and swallowed air.[4]

Fluid loss from emesis, bowel oedema and loss of absorptive capacity leads to dehydration. Emesis leads to loss of gastric potassium, hydrogen and chloride ions, and significant dehydration stimulates renal proximal tubule reabsorption of bicarbonate and loss of chloride, perpetuating the metabolic alkalosis.[5] In addition to derange-ments in fluid and electrolyte balance, intestinal stasis leads to overgrowth of intestinal flora, which may lead to the development of feculent emesis. Additionally, overgrowth of intestinal flora in the small bowel leads to bacterial translocation across the bowel wall.[6] Ongoing dilation of the intestine increases luminal pressures. When luminal pressures exceed venous pressures, loss of venous drainage causes increasing edema and hyperemia of the bowel. This may eventually lead to compromised arterial flow to the bowel, causing ischaemia, necrosis and perforation. A closed-loop obstruction may undergo this process rapidly. Intestinal volvulus, the prototypical closed-loop obstruction, causes torsion of arterial inflow and venous drainage, and is a surgical emergency.[7, 8]

More severe or prolonged under perfusion of kidney due to hypovolaemia, shock may lead to failure of compensatory mechanism and hence an acute decline in GFR. This may lead to formation of low volume of urine or anuria. Ischaemia and toxic insults to the kidney preferentially cause cell death of tubular epithelial cells, promoting decreased kidney function.[9, 10]

As the P^H decreases (<7.35), it implies acidosis, while if the P^H increases (>7.45) it implies alkalosis. In the context of arterial blood gases, the most common occurrence will be that of respiratory acidosis. Carbon dioxide is dissolved in the blood as carbonic acid, a weak acid; however, in large concentrations, it can affect the P^H drastically. Whenever there is poor pulmonary ventilation, the carbon dioxide levels in the blood are expected to rise. This leads to a rise of carbonic acid, leading to a decrease in P^H. The first buffer of P^H will be the plasma proteins, since these can accept some H^+ ions to try and maintain homeostasis. As carbon dioxide concentrations continue to increase ($PaCO_2$ > 45 mmHg), the condition is known as respiratory acidosis. The body tries to maintain homeostasis by increasing the respiratory rate. This allows much more carbon dioxide to escape the body through the lungs, thus increasing the P^H by having less carbonic acid. If a patient is in a critical setting and intubated, one must increase the number of breaths mechanically.

On the other hand, respiratory alkalosis ($PaCO_2$ < 35mmHg) occurs when there is too little carbon dioxide in the blood. This may be due to hyperventilation or else excessive breaths given via a mechanical ventilator in a critical care setting. The action to be taken is to calm the patient and try to reduce the number of breaths being taken to normalize the P^H. The respiratory pathway tries to compensate for the change in P^H

in a matter of 2–4 hours. If this is not enough, the metabolic pathway takes place.

The kidney and the liver are two main organs responsible for the metabolic homeostasis of P^H. Bicarbonate is a base that helps to accept excess hydrogen ions whenever there is acidaemia. However, this mechanism is slower than the respiratory pathway and may take from a few hours to 3 days to take effect. In acidaemia, the bicarbonate levels rise, so that they can neutralize the excess acid, while the contrary happens when there is alkalaemia. Thus when an arterial blood gas test reveals, for example, elevated bicarbonate, the problem has been present for a couple of days, and metabolic compensation took place over a blood acidaemia problem.

In general, it is much easier to correct acute P^H derangements by adjusting respiration. Metabolic compensations take place at a much later stage. However, in a critical setting, a patient with a normal P^H, high CO_2 and high bicarbonate means that, although there is a high carbon dioxide level, there is metabolic compensation. As a result one must be careful as to not artificially adjust breaths to lower the carbon dioxide. In such case, lowering the carbon dioxide abruptly means that the bicarbonate will be in excess and will cause a metabolic alkalosis. In such a case, carbon dioxide levels should be slowly diminished.[11]

METHODS

This was a cross-sectional observational study was carried out among 200 subjects suffering from acute and acute-on-chronic intestinal obstruction to observe the electrolytes and blood gas changes. The study was carried out in different surgical wards of Dhaka Medical College, Dhaka during July – December' 2013. The study subjects were enrolled after fulfillment of the inclusion criteria. They were collected from the referred patients attending in out-patient department of surgery and also from in-patient department of the respective discipline. Blood was collected immediately after admission (before resuscitation) and after resuscitation of the patients those arrived in hospital with early or delayed onset of symptoms. Blood samples were collected from radial or femoral artery with aseptic and necessary precaution in the Intensive Care Unit (ICU). Then the findings of the different samples were compared among the acute and acute-on-chronic intestinal obstruction cases.

RESULTS

Among total 200 cases of intestinal obstruction, 110 were acute intestinal obstruction and 90 were acute-on-chronic intestinal obstruction. Age range of patients was 18-65 years. The highest number (35, 32%) of age group was 31-40 years in acute intestinal obstruction and that in acute-on-chronic obstruction was 41-50 years age groups (30, 33.3%). In acute obstruction, 70 (63.7%) were male and 40 (36.3%) were female and in acute-on-chronic obstruction group, 62 (68.8%) were male and 28 (31.2%) were female (Table I).

Table I: Demographic characteristics of patients (n-200)

Demographic variables		Acute intestinal obstruction (n= 110) Number (%)	Acute-on-chronic obstruction (n=90) Number (%)
Gender	Male	70 (63.7)	62 (68.8)
	Female	40 (36.3)	28 (31.2)
Age group in years	18- 30	25 (22.7)	10 (11.1)
	31-40	35 (32)	15 (16.7)
	41-50	20 (18.2)	30 (33.3)
	51-60	20 (18.2)	25 (27.8)
	>61	10 (9)	10 (11.1)

Common causes of intestinal obstruction in this study were adhesion 64 (32%), intestinal TB 57 (28.5%), malignancy 24 (12%), hernia 23 (11.5%), volvulus 18 (9%) (Table II).

Table II: Causes of Intestinal Obstruction

Causes of obstruction	n (%)
Adhesion	64 (32%)
Intestinal tuberculosis(TB)	57 (28.5%)
Malignancies	24 (12%)
Hernia	23 (11.5%)
Volvulus	18 (9%)
Worms	08 (4%)
Faecal impaction	06 (3%)
Total	**200 (100%)**

Most subjects with acute intestinal obstruction had hyponatraemia (69.0%), 72.7% hypokalaemia, 70.9% hypochloraemia and 67.3% hypocalcaemia before resuscitation while 83.6% hyponatraemia, 80.9% hypokalaemia, 81.8% hypochloraemia and 80% hypocalcaemia were corrected after resuscitation (Table III).

Table III: Electrolytes change in acute intestinal obstruction

Electrolytes	Number (%) of acute intestinal obstruction showing electrolytes changes					
	On admission (%)			After resuscitation (%)		
	Normal	Below normal	Above normal	Normal	Below normal	Above normal
Na^+	16 (14.5)	76 (69.0)	18 (16.4)	92 (83.6)	8 (7.3)	10 (9.0)
K^+	17 (15.4)	80 (72.7)	13 (11.8)	89 (80.9)	14 (12.7)	7 (6.4)
Cl^-	14 (12.7)	78 (70.9)	18 (16.5)	91 (81.8)	9 (8.2)	10 (9.0)
Ca^{++}	18 (16.4)	74 (67.3)	18 (16.5)	88 (80.0)	13 (11.8)	9 (8.2)

Table IV shows that, majority of the subjects had alkalosis (70.9%) and decreased $PaCO_2$ (70.9%) before resuscitation. After resuscitation, P^H (80.9%) and $PaCO_2$ (78%) returned to normal.

Table IV: Blood gas changes in acute intestinal obstruction

Blood gases	Number (%) of acute intestinal obstruction showing blood gas changes					
	On admission (%)			After resuscitation (%)		
	Normal	Below normal	Above normal	Normal	Below normal	Above normal
P^H	18 (16.3)	14 (12.7)	78 (70.9)	89 (80.9)	14 (12.7)	7 (6.4)
$PaCO_2$	18 (16.3)	78 (70.9)	14 (12.7)	87 (78.0)	15 (13.6)	9 (8.2)
PaO_2	84(76.4)	26 (33.6)	00	94 (85.4)	14 (12.5)	2 (2.0)
HCO_3	20 (18.1)	14 (12.7)	76 (69.9)	85 (77.3)	17 (15.4)	8 (7.3)

In acute-on-chronic intestinal obstruction, most subjects (78.9%) had hyponatraemia, 82.2% hypokalaemia, 77.8% hypochloraemia and 81.1% hypocalcaemia before resuscitation while 77.8% hyponatraemia, 75.5% hypokalaemia, 80% hypochloraemia and 77.8% hypocalcaemia was corrected after resuscitation (Table V).

Table V: Electrolytes change in acute-on-chronic intestinal obstruction

Electrolytes	Number (%) of acute-on-chronic intestinal obstruction showing electrolytes changes					
	On admission (%)			After resuscitation (%)		
	Normal	Below normal	Above normal	Normal	Below normal	Above normal
Na^+	10 (11.1)	71 (78.9)	09 (10.0)	70 (77.8)	14 (15.5)	06 (6.7)
K^+	06 (6.7)	74 (82.2)	10 (11.1)	68 (75.5)	13 (14.4)	09 (10.0)
Cl^-	11 (12.2)	70 (77.8)	09 (10.0)	72 (80.0)	12 (13.3)	06 (6.7)
Ca^{++}	07 (7.8)	73 (81.1)	10 (11.1)	70 (77.8)	14 (15.5)	06 (6.7)

Among these patients, majority of them had alkalosis (74.4%) and decreased $PaCO_2$ (73.3%) before resuscitation. After resuscitation, P^H (75.5%) and $PaCO_2$ (73.3%) returned to normal (Table VI).

Table VI: Blood gas changes in acute-on-chronic Intestinal Obstruction

Blood gases	Number (%) of acute-on-chronic intestinal obstruction showing blood gas changes					
	On admission (%)			After resuscitation (%)		
	Normal	Below normal	Above normal	Normal	Below normal	Above normal
P^H	07 (7.8)	16 (17.8)	67 (74.4)	68 (75.5)	14 (15.5)	08 (8.9)
$PaCO_2$	10 (11.1)	66 (73.3)	14 (15.5)	66 (73.3)	15 (16.7)	09 (10.0)
PaO_2	64 (71.1)	26 (28.9)	00	81 (90.0)	09 (10.0)	00
HCO_3^-	10 (11.1)	16 (17.8)	64 (71.1)	70 (77.8)	14 (15.5)	06 (6.7)

DISCUSSION

Nowadays, acute and acute-on-chronic intestinal obstructions remain the most serious common cause for emergency laparotomy. Although the mortality rate continues to decrease with a better understanding of the pathophysiology, improvement of diagnostic techniques and greater stress on correction of fluid and electrolyte imbalance, most of these are limited to developed countries or the major centers in other countries.[12,13] Previous study revealed that adhesions are the single most common cause for small bowel obstruction. Non adhesive aetiologies of bowel obstruction include incarcerated hernias, obstructive lesions (malignant and benign), and a number of infrequent causes for bowel obstruction such as bezoars, inflammatory bowel disease, and volvulus.[14] The causes of SBO in pediatric patients include intussusceptions, congenital atresia and stenosis.[15] However, in present study, causes of intestinal obstruction were adhesion (32%), intestinal TB (28.5%), malignancy (12%), hernia (11.5%), volvulus (9%), worms (4%) and faecal impaction (3%).

Due to repeated vomiting in acute and acute-on-chronic obstruction, there is loss of sodium and potassium with gastric acid loss resulting in hyponatraemia, hypokalaemia and metabolic alkalosis.[3,4,5] On admission, these patients usually found collapsed due to excessive fluid loss which is resulting electrolytes imbalance and altered arterial blood gas level.[6,7,8]

In current study, most of the subjects with acute intestinal obstruction (69.1%) had hyponatraemia and 72.8% hypokalaemia before resuscitation. Another study shown that, serum sodium levels were ranged from 121 mEq/L to 133.3 mEq/L. Persistant hyponatremia with no relation to duration of obstruction and no changes in serum potassium level were noted.[16]

Any type of obstruction is first treated conservatively, followed by investigation to find out cause of obstruction than definitive management by laparotomy. Mortality and morbidity are dependent on the early recognition, correct diagnosis of obstruction and proper surgical intervension.[17, 18]

Hyponatraemia is a common electrolyte disorder among hospitalized patients and has been associated with increased mortality. The goal is to raise the serum sodium level by 1.5 to 2mEq/L/hour until symptoms subside or until the concentration has risen to a safer level-- usually greater than 118 to 120mEq/L, with the primary focus being to minimize the risk of seizures.[19]

CONCLUSION

In this study, it was seen that both acute and acute-on-chronic intestinal obstruction patients had hyponatraemia, hypokalaemia and abnormal ABG. The majority of the abnormalities of ABG were corrected in both conditions. But the correction of electrolytes imbalance (hyponatraemia and hypokalaemia) is relatively more in acute obstruction than in acute-on-chronic obstruction which influences the better surgical outcome in acute intestinal obstruction.

Conflicts of Interest: None

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