

ELECTROLYTE IMBALANCES IN ACUTE INTESTINAL OBSTRUCTION IN ADULTS

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ABSTRACT

Introduction: Electrolytes are salts in the body that conduct electricity. Proper balance is essential for muscle coordination, heart function, fluid absorption and excretion. Bowel obstruction describes failure of aboral progression of intestinal contents. **Aims and objectives:** The aim of present study is to determine the electrolyte imbalances in acute intestinal obstruction in adults presenting with acute abdomen. **Materials and methods:** The material for this dissertation includes 35 cases of acute intestinal obstruction in adult patients, operated in Darbhanga Medical College and Hospital, Laheriasarai, Bihar. The present study comprises of cases admitted during one year. Emphasis was placed upon determining the exact duration of obstruction and previous treatment including fluid therapy received prior to admission. Before starting therapy blood sample was taken for investigation and analysis. **Results & Discussion:** The percentage of surgical admission reads 1.01%. *Adhesive obstructions especially post operative* formed the largest group in the series. Most of the obstructions were *simple* (57.14%). 37.14% patients presented with gangrene and 5.17% had perforation and peritonitis. Individual *serum sodium* levels in this series ranged from 121 mEq/L to 133.3 mEq/L. Mean potassium values ranged from 3.9-5.1 mEq/L. Serum chloride ranged from 90.5 to 98.2 mEq/L. **Conclusion:** The prognosis of intestinal obstruction can be considerably improved when the extent of associated water and electrolyte loss and need for intravenous replacement can be recognized.

KEYWORDS: Intestinal obstruction, electrolyte imbalances, acute obstruction.

INTRODUCTION

Electrolytes are salts in the body that conduct electricity. They are found in the body fluid tissue and blood. Examples are chloride, calcium, magnesium, sodium and potassium. Sodium is concentrated in extracellular fluid, potassium is concentrated in intracellular fluid. Proper balance is essential for muscle coordination heart function, fluid absorption and excretion, nerve function and concentration.

Bowel obstruction describes failure of aboral progression of intestinal contents. Based on nature, severity, location and etiology several terms are used to describe bowel obstruction e.g. functional, mechanical etc. *Simple mechanical* obstruction is compromise of lumen of bowel without compromise of its vascular supply. In simple obstruction, important and progressive changes take place in the bacteriologic content of obstructed bowel, in the amount and composition of gas in the gut above obstruction. When *strangulation* complicates the picture, these pathological changes are compounded by the progressive vascular changes in the affected intestine and its mesentery and eventually lead to toxemia associated with actual death of gut wall.

REVIEW OF LITERATURE

Baddhagudodhara the Sanskrit name for intestinal obstruction was mentioned in chapter 7 of Sasrutha - Samhitha. He goes on to describe that "when food or sticky substance gathers collectively or separately in intestine of patients, the faeces along with food gradually accumulate in him like that in a drain. It distends his abdomen between precardium and umbilicus, faecal smell is present in vomitus - this condition should be known as 'baddhagudodhara'. In early 1900 proctoclysis was used to support patient who were unable to take fluids by mouth.

Fluid and electrolyte imbalance in intestinal obstruction/ Pathophysiology of Intestinal obstruction

Studies conducted by David Sung and Williams^[1] in experimental intestinal obstruction in mongrel dog revealed that there is accumulation of fluid due to decreased absorption and increased secretion into the lumen. These experiments brought into notice that rapid rehydration with hypotonic fluid caused influx of more fluid into the obstructed gut thus worsening the condition or hastening changes of strangulation.

Shields^[2] studied the handling of water and electrolytes by obstructed gut. His result also confirmed that within 12 hours of obstruction, ileum above the site ceased to absorb and began to secrete water. This process increased rapidly over 48 hours. The absorption was reduced to one third its original rate in 60 hours. He also mentioned that 1-8 meq of sodium accumulated in lumen of ileal segment in 60 hrs. The longer the obstruction persisted the greater became the potassium secretion. The rate was 0.09 mEq/every 10 minutes after 6 hours. If entire small bowel responded in likely manner, 1.4 litres of fluid would be lost into the intestinal lumen every 4 hours. The initial losses are thus isotonic and thus not much of imbalance occur in the initial stages.

Eggleston^[3] et al noted that there is a persistent hyponatremia, a gradual drop in serum potassium beginning 5 days after onset of obstruction, a progressive decrease in chloride level in first week followed by a rise, a progressive rise in urea level for week and an initial tendency towards acidosis gradually replaced by alkalosis.

The epithelial cells of intestinal mucosa which are joined at their luminal border by tight junction are freely permeable to water and less permeable to ions. In intestinal obstruction there is impairment of venous flow due to distention leading to increased capillary pressure with consequent diffusion of water and electrolyte into intracellular spaces which causes increased pressure on tight junctions. This results in flow of isomolar fluid with sodium concentration near that of plasma into the lumen.

Potassium losses are not seen in initial days due to immense stores of intracellular potassium. Excessive urinary loss of potassium without normal dietary intake is more important than loss into gut lumen as a cause of hypokalemia in intestinal obstruction. Hypokalemia results from.

1. Alkalosis
2. Hypochloremia.
3. Hyperaldosteronism which is due to hyponatremia.

In alkalosis there is increased tubular fluid pH, due to increased bicarbonate in tubular fluid. This causes increased permeability of luminal membrane to potassium leading to increased potassium excretion. Since the cellular reservoir of potassium is large, deficiencies are not seen in initial period. Thus with adequate rehydration potassium levels return back of normal. To compensate for chloride loss from vomiting, bicarbonate diffuses into the extracellular fluid leading to hypochloremia and metabolic alkalosis. In small bowel, the cells pump hydrogen into the extracellular fluid and converts bicarbonate to carbon dioxide and water, the bicarbonate formed in cells diffuse into the lumen and net effect is loss of bicarbonate from body leading to metabolic acidosis.

Restoration of Fluid and Electrolyte balance

Diagnosis of fluid electrolyte disturbance and management of this condition is the mainstay of treatment in patients with intestinal obstruction. Repeated physical examination aided by frequent acid base status forms part of this management.

1. In mild dehydration patient has lost 4% of body weight and complains of thirst e.g : $70\text{kg} \times 0.04 = 2.8$ liter deficit. In moderate dehydration (marked thirst dry mucous membrane, absent sweating, loss of skin texture), 6% of body water is lost and in severe dehydration 10% of total body water is lost. With marked dehydration, hypotension etc. patient may be confused or delirious.
2. In hypernatremia, if patient has water deficit it is calculated as follows given that normal serum sodium is 140meq and total body water is 60% of body weight ($0.6 \times \text{body weight in kg}$).
 $\text{Water deficit} = (\text{Observed sodium} / 140) \times 0.6 \times \text{body weight in kg}$.
3. Electrolyte deficits like sodium, chloride and bicarbonate can also be calculated.
 $\text{Deficit} = (\text{normal volume} - \text{observed volume}) \times \text{electrolyte distribution} \times \text{body weight}$ (here sodium distribution is 60%, chloride is 20% and bicarbonate is 50%).
4. Potassium deficits cannot be calculated with normal blood pH, the estimate is for every 1 meq decrease in potassium concentration between normal and 3 meq/L. Consider total body deficits as 100-200 meq/L. For every 1.0 meq/L decrease in potassium concentration below 3.0meq/L, consider total body deficit as 300-400 mEq/L. The concentration of infusion should not exceed 40 meq/hour or 200 meq/day (or 0.2 meq/kg/hour).

Key Factors

- 1- Emphasis must be placed on determining duration of obstruction, since this fact is important in treatment as reflected in the varying electrolyte values (Eggleston et al)^[3]
- 2- It must be remembered that volume for volume replacement of losses is more important since renal mechanism, if intact, will take care of fluctuating electrolyte concentration.
- 3- Since the losses are isotonic, it should be replaced by isotonic solution (normal saline, ringer lactate) and not by hypotonic solution. Massive losses will need replacement of potassium.
- 4- In spite of having number of formulas for correcting various deficits correct clinical assessment and measurement of losses with volume to volume replacement still holds good as mainstay of treatment.
- 5- Other than correcting visible losses and providing good urine flow maintenance of nutrition, replacement of blood loss, if any (strangulation) and protein balance are also important since nutritional defect is directly proportional to the duration of obstruction.

6- Failure to restore circulatory stability will almost invariably result in an operative mortality.

AIMS AND OBJECTIVES

The aim of present study is to determine the electrolyte imbalances in acute intestinal obstruction in adults presenting with acute abdomen in the emergency department of Darbhanga Medical College and Hospital, Laheriasarai, Bihar, India.

MATERIALS AND METHODS

The material for this dissertation includes 35 cases of acute intestinal obstruction in adult patients, operated in Darbhanga Medical college and Hospital, Laheriasarai, Bihar. The present study comprises of cases admitted during one year.

Inclusion criteria

1. Acute cases treated surgically.
2. Diagnosis of obstruction proved at time of operation. *Following were excluded in the study.*
- 1- Patients with external hernia in which only omentum was found.

2. *Duration of symptoms prior to admission and associated mortality.*

Duration in days	No of cases	Percentage	Deaths	Mortality
1 day	4	11.4	2	50%
2 days	7	20	-	-
3 days	3	8.5	-	
4 days	7	20	-	
5 days	5	14.2	-	
6 days	4	11.4	1	25%
7 days	3	8.5	-	
>7 days	2	5.7	-	
Total	35		3	8.5%

3. *Distribution of causes by etiology.*

Causes	No of cases	Percentage (%)
Adhesions Postoperative – 15 Inflammatory – 1 Congenital band – 2	18	51.4
External Hernia	9	25.7
Strictures	3	8.5
Malignancy	1	2.8
Intussusceptions	3	8.5
Internal Hernia	1	2.8
Total	35	

4 *Classification by intraoperative pathology*

Pathology	Cases	% of total	Deaths	Mortality
Simple	20	57.14%	-	
Obstruction				
Gangrene	13	37.14%	1	7.6%
Perforation	2	5.71%	2	100
Peritonitis				

2- Patient with adynamic or post operative ileus.

Emphasis was placed upon determining the exact duration of obstruction and previous treatment including fluid therapy received prior to admission. Before starting therapy blood sample was taken for investigation and analysis.

RESULTS AND ANALYSIS

1. Incidence

Incidence of acute intestinal obstruction in Darbhanga Medical College & Hospital, Laheriasarai, Bihar in one year period.

Total number of admission in surgical ward in this period including all types of acute abdomen was 3445 and total number of acute intestinal obstruction operated during this period was 35. The data for total number of acute intestinal obstruction admitted was not available. The percentage of surgical admission thus reads 1.01%. There was a male predominance due to inclusion of inguinal hernias. The oldest patient was 75 year old and youngest patient was 15 yrs old.

5 Electrolyte imbalances

Duration	Serum sodium changes	Serum potassium changes	Serum chloride changes	Serum bicarbonate changes	Serum urea changes
1 day	129.7	4.15	88.9	20.5	52
2 days	129.7	4.3	96.8	20.6	35.2
3 days	133.3	5.1	92.6	20.6	42.6
4 days	128.5	3.9	92.1	19.7	51.7
5 days	124.0	4.26	92.2	21.6	58.3
6 days	121.0	4.5	87	18.5	68.0
7 days or more	129.0	4.7	90	22	43.6

DISCUSSION

The 35 cases of acute intestinal obstruction in adults operated in Darbhanga Medical College and Hospital, Laheriasarai, formed the basis of this study. Most of patients presented between 2nd and 5th day. The longest duration of presentation was 10 days. There were 3 deaths in the group (8.5%). With delayed presentation the mortality and morbidity was expected to be high, but this was not so in the case, since patients who presented late in our hospital had already received treatment from outside and hence were relatively in stable conditions.

Adhesive obstructions especially post operative formed the largest group in this series (51.4%). The incidence of external hernia was less (25.7%). This could be because hernias are usually treated before onset of any complications. 3 cases of hernia presented with strangulation (33.33%). The increase in adhesions as a cause of obstruction can be attributed to the increase in frequency of abdominal surgery performed. Post operative adhesions formed 83% of causes for adhesive obstruction. But Wagensteen^[4] reported 31% as the incidence. Eggleston reported the incidence of adhesions and hernia as 15 and 27 percent.

Most of the obstructions were *simple* (57.14%). 37.14% patients presented with gangrene and 5.17% had perforation and peritonitis. Strangulation in external hernia was noted in 33.33% patients. Nyhus reported 24% cases of strangulation due to hernias. Patients who developed perforation had 100% mortality in spite of early presentation. The cause of death in these patients was multifactorial including electrolyte imbalance and septicaemia.

From the above observation it can be summarized that incidence of intestinal obstruction due to adhesions are on the rise and external hernias have fallen to second place (Ellis H). The incidence of strangulated hernia has also fallen due to the prophylactic benefit of elective surgery.

Death rate for intestinal obstruction had fallen from 50 per million in 1940 to 30 per million in 1950. Even through decline in rates continue it is less sharp than before. Compared to 1920's and 30's where mortality rates were 26% the present mortality is in range of 15%.

Ellis in 1980 reported mortality of 6%. Eggleston reported 16.2% (1972). The improvement in prognosis of intestinal obstruction was attributed to improved anaesthesia techniques, better knowledge of fluid and electrolyte replacement, efficient blood transfusion services and introduction of antibiotics. A longer segment, perforation delay in treatment with severe distention and gross electrolyte and fluid imbalance and extreme of age are major factors influencing survival rate. The patients who presented with perforation had 100% mortality and patients with strangulation had 7.6% mortality. The improved results especially with relation to strangulation could be attributed to early diagnosis and treatment before presenting to our hospital.

Electrolyte imbalance

The prognosis of intestinal obstruction was considerably improved when the extent of associated water and electrolyte loss and need for intravenous replacement were recognized (Aird 1941; Hartwell and hognet 1912). It is also realized that the composition and quantity of infused must be carefully controlled if much is not to be wasted by loss into intestinal lumen and urine. The increased intestinal loss, an obviously undesirable side effect is not always appreciated. The *objective* of this study was thus to highlight the importance of knowing the fluid and electrolyte disturbances in patients with intestinal obstruction and correlation of these deficits by intravenous infusion so that deficits are not only corrected but replaced fluid is distributed correctly between body compartments, thus improving the prognosis of that patient.

Individual *serum sodium* levels in this series ranged from 121 mEq/L to 133.3 mEq/L (Normal 132-144 mEq/L). In Eggleston series the values ranged from 130.5 to 138.7 mEq/L. Thus a persistent hyponatremia with no relation to duration of obstruction was noted in both series. Eggleston explains that this could be because of two factors-first, a mild dilutional effect of ingestion of relatively salt free fluid in early phase of obstruction and second (most important) ionic shifts occurring at cellular level. The increase in cell membrane permeability results in increase rate of influx of sodium and water. If sodium pump removes the excess, then intracellular sodium remains normal or else there is accumulation of sodium and chloride. This leads to hyponatremia. Water also moves into the cell because of

shift of sodium. Intestinal obstruction also causes decreased absorption of electrolytes from lumen (hyponatremia). If enough sodium and water are available by iso-osmolar replacement at this stage the imbalances can be corrected.

A gradual drop in *serum potassium* level beginning 5 days after obstruction was noted in Egglestone series. No such changes were noticed in this series. Mean values ranged from 3.9-5.1 (normal 3.5-5.3 mEq/L). Significant total body potassium depletion would only occur after loss of many litres of intestinal fluid. Moreover the immense body store of potassium will maintain the serum levels to normal. Thus adequate rehydration, without supplements itself can correct potassium levels. Eggleston et al noticed drop in serum potassium after 4-5 days of obstruction reflecting depletion in intracellular store of potassium.

Normal *serum chloride* ranges from 95-107 mEq/L. In this series serum chloride ranged from 90.5 to 98.2 mEq/L. In Eggleston series a progressive decrease in chloride level over the 1st week followed by a rise was seen. Elevation of blood *urea levels* especially after 3rd day of obstruction.

SUMMARY AND CONCLUSION

1. A review of 35 cases of intestinal obstruction in adults operated during one year was presented with emphasis on electrolyte imbalance. Emphasis was placed on determining the exact duration of obstruction and previous treatment including fluid therapy received prior to admission.
2. Intestinal obstruction formed 1.01% of surgical admission.
3. Treatment received prior to admission was of significance with relation to the prognosis and decrease of mortality rates.
4. Adhesions especially postoperative adhesions formed the major bulk of admissions. External hernia stood second in the list. The former as a result of community becoming more surgically sophisticated and the later due to prophylactic benefit of elective surgery.
5. Complications of obstruction are associated with more mortality.
6. The improved mortality and morbidity rates could be attributed to early diagnosis and treatment before presenting to hospital in spite of presenting late to hospital.
7. A persistent hyponatremia, remarkably constant serum potassium, hypochloremia, a rise in urea levels and acidosis were noted. Comparisons have been made with the available series.
8. Shock, dehydration, electrolyte changes and septicemia are important causes of death.
9. Better prognosis is attributed to better anaesthetic techniques, better knowledge of fluid and electrolyte replacement, efficient blood transfusion services and

introduction of antibiotics and gastrodueodenal suction.

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