Original Article

Electrolyte Imbalance in Admitted Diabetic Patients: Patterns and Factors Responsible- A Cross Sectional Study

Ahmed SS¹, Nur F², Ullah MR³, Mamun AA⁴, Laila TR⁵, Khan MAM⁶, Ali MZ⁷

Abstract

Diabetes Mellitus (DM) is a major burden upon health-care facilities in all countries. Electrolyte imbalances (EI) are very common in admitted patients with diabetes which leads to substantial morbidity and mortality. Every patient with diabetes needs thorough evaluation for the etiology and precipitating factors responsible for EI and therefore rational management. Data regarding the incidence of EI in DM in our country is limited. There is no existing record for profiling of EI in Bangladeshi diabetic subjects. The objective of this study was to find out the common types of EI in hospitalized diabetic patients and to determine the influencing factors responsible for them. Samples were selected from all adult nonpregnant diabetic patients admitted in the indoor, department of Medicine with electrolyte disturbances irrespective of their cause of admission. A total number of 150 admitted diabetic patients with electrolyte imbalance were interviewed, examined physically and laboratory specimen taken for biochemical analysis. Information was collected in a semi structured data collection form for analysis. Hyponatremia was found to be the most common EI in admitted diabetic patients. Electrolyte imbalance was found to be more common in the aged and female populations. Common factors influencing EI were vomiting, diarrhea, sweating, uncontrolled diabetes with severe hyperglycemia and certain drugs especially diuretics. Among the other well established complications of diabetes like acute metabolic complications, microangiopathy and macroangiopathy, EI should also be considered especially in the admitted patients.

Keywords: Electrolyte imbalance, Diabetes, Hyponatremia, Hypokalemia.

Introduction

Background: Electrolyte imbalance in diabetes

Electrolyte imbalances are common in DM, and many patients develop acute EI warranting hospital admission.

Some diabetic patients are found to have EI while admission for reasons like severe hyperglycemia or acute metabolic complications like diabetic ketoacidosis. Treatment of EI involves first determining

- 1. Prof. (Dr.) Sheikh Salahuddin Ahmed, Head, Department of Internal Medicine, Bangladesh Institute of Health Sciences (BIHS) & Hospital, Dhaka, Bangladesh.
- 2. Dr. Fazle Nur, Consultant, Department of Endocrinology, BIHS & Hospital, Dhaka, Bangladesh.
- 3. Dr. Md. Rahamat Ullah, Registrar, Department of Medicine, BIHS & Hospital, Dhaka, Bangladesh.
- 4. Dr. Abdullah Al Mamun, Medical Officer, Department of Medicine, BIHS & Hospital, Dhaka, Bangladesh.
- 5. Dr. Tarafdar Runa Laila, Assistant Professor, Department of Obstetrics & Gynecology, Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka, Bangladesh.
- 6. Dr. Md. Abdul Mahid Khan, Assistant Professor, Department of Cardiology, BIHS & Hospital, Dhaka, Bangladesh.
- 7. Prof. (Dr.) Md. Zulfikar Ali, Head, Department of Medicine, Khwaja Yunus Ali Medical College & Hospital, Enayetpur, Enayetpur Sharif, Sirajgonj, Bangladesh.

Correspondence:

Prof. (Dr.) Sheikh Salahuddin Ahmed, email: drsksalahahmed@yahoo.com, Mobile: 01711-568169

cause and then correcting this when possible. Data regarding the incidence of EI in DM in our country is limited. Electrolyte imbalance in diabetes is primarily a result of deficiency of insulin and elevated blood glucose. Other common causes are vomiting, fever, excessive sweating, and diarrhea. A low serum Na+ concentration or hyponatremia is the most common and important electrolyte abnormality encountered in hospitalized patient¹. Others are hypokalemia, hypomagnesemia and hyperkalemia.

These abnormalities are common in older adults, and increasing age is associated with a marked increase in the incidence of hypokalemia². Both hyponatremia and hypokalemia can cause substantial morbidity and mortality, and ironically, incorrect treatment can add to the problem. Potassium facilitates the function of insulin in the delivery of glucose to cells; when insulin binds to its receptors on the cell membrane, it causes K+ to flow into the cells. Therefore in diabetics, excessive use of human insulin may be associated with hypokalemia.

It is well known that the hyperglycemia in DM can cause osmotic diuresis leading to dehydration and electrolyte loss, particularly of Na+and K+. Dehydration in turn induces secondary hyperaldosteronism that exacerbates K+ loss. As a result patient with DM may have hyponatremia and likely to develop hypokalemia³. Osmotic diuresis resulting from hyperglycemia in DM in addition to Na+ and K+, may promote net loss of other minerals and electrolytes, including Mg++, calcium (Ca++), and chloride⁴⁻⁶. In patient with diabetic ketoacidosis, hydrogen (H+) ion is elevated in the blood and the resulting metabolic acidosis forces H+ into cells, displacing K+ out of the intracellular compartment. Plasma K+ may even be raised initially; however, soon after insulin treatment is started there is likely to be a precipitous fall in the plasma K+ due to the movement of K+ into cells induced by insulin and a patient is prone to develop hypokalemia. A patient with hyperosmolar nonketotic diabetic patient can also present with EI.

Diabetic nephropathy is one of the complications of DM, which ultimately leads to renal failure and renal failure may be a cause of EI among hospitalized diabetic patients. Example of drugs used for treating comorbid conditions like hypertension in diabetes are thiazide or loop diuretics, Angiotensin Converting Enzyme Inhibitors (ACEIs) and Angiotensin 2 Receptor Blockers (ARBs) which may also result in EI. Furthermore insulin secretion is decreased in hypokalemia^{7,8}, and K+

infusions improve glucose tolerance by increasing insulin and proinsulin concentrations⁹.

Hyponatremia

Hyponatremia is defined as a serum Na+ concentration less than 135 mmol/L1,¹⁰⁻¹³. Patients with clinically significant hyponatremia present with nonspecific or neurologic symptoms attributable to cerebral edema. Hypertonic hyponatremia is usually due to hyperglycemia. Isotonic hyponatremia may occur in conditions like hyperlipidemia and hyperproteinemia. In general, hypotonic hyponatremia occurs due either to (i) a primary Na+ loss (Na+ deficit with a relatively smaller water deficit) like sweating, gastrointestinal loss: vomiting, diarrhea; renal loss: diuretics, salt wasting nephropathy; or (ii) due to a primary water gain (water retention alone) like Syndrome of Inappropriate ADH (SIADH), hypothyroidism, primary polydipsia; or (iii) due to a primary Na+ gain (Na+ retention with relatively greater water retention) like heart failure, hepatic cirrhosis, nephrotic syndrome & chronic renal failure. It is important to note that cerebral salt wasting factors liberated after neurosurgery are also the cause of hyponatraemia¹².

Hypokalemia

Hypokalemia is defined as a serum K+ concentration less than 3.5 mmol/L. It may result from one or more of the following mechanisms: (i) inadequate intake of K+ but is unlikely to be the only cause; (ii) shift into cells like metabolic alkalosis, insulin, adrenergic agonist; and (ii) increased net loss generally, from the body, through either the gastrointestinal tract or the kidney like vomiting, diarrhea, diuretics, primary and secondary hyperaldosteronism. The most frequent cause of excess renal K+ loss is metabolic alkalosis following Cl- loss secondary to vomiting^{14,15}. However, dietary K+ restriction may exacerbate the hypokalemia secondary to increased gastrointestinal or renal loss¹².

Hypomagnesemia

The concentration of Mg++ in serum is closely regulated within the range of $0.75-1.0 \text{ mmol/L}^{13}$. Reduced intake alone is rarely sufficient to cause Mg++ depletion, and generally there is excessive loss from either the gastrointestinal tract (notably in chronic diarrhea, prolonged vomiting/nasogastric aspiration, laxative abuse, malabsorption, small bowel bypass surgery) or the kidney (during prolonged use of loop diuretics). Excessive alcohol ingestion can cause Mg++ depletion through both gut and renal losses¹⁶.

Hyperkalemia

Hyperkalemia defined as plasma K+ concentration >5.1 mmol/L, occurs as a result of either K+ release from cells or decreased renal loss¹³. Increased oral K+ intake is rarely the sole cause of hyperkalemia, since the phenomenon of K+ adaptation ensures rapid K+ excretion in response to increase in dietary consumption. Insulin deficiency and hypertonicity due to hyperglycemia promote K+ shift from the intracellular fluid to extracellular fluid. ACEIs, ARBs and spironolactone used for congestive cardiac failure (CCF), hypertension or chronic kidney disease (CKD) are often responsible for hyperkalemia.

Objectives

- 1. To find out the type of EI in diabetic patients admitted in BIHS & Hospital.
- 2. To identify the factors responsible for EI in diabetic patients.
- 3. To find out the comorbidities associated with DM.
- 4. To assess the demographic status of the diabetic patients admitted in BIHS & Hospital and assess their effect on EI.

Rationale

Diabetes is a major burden upon health-care facilities in all countries³. The incidence and prevalence of diabetes is increasing specially in the aged populations³. It remains a leading cause of cardiovascular disorders, blindness, end-stage renal failure, amputations and hospitalizations¹⁷. Electrolyte imbalances are very common in admitted patients with DM which causes substantial morbidity and mortality. The incidence of electrolyte disturbance is much more in the elderly mainly owing to impaired ability to maintain water and electrolyte homeostasis in response to dietary and environmental changes¹⁸. Data regarding the incidence of EI in DM in our country is limited. There is no existing record for profiling of electrolyte imbalance in Bangladeshi diabetic subjects. The well established complications of diabetes as described in standard books are acute metabolic, microvascular and macrovascular complications³. But we also face electrolyte disturbances routinely in our clinical practice so EI should also be considered as an important complication especially in the diabetic patients admitted in hospital. Therefore serum electrolytes should be checked routinely in hospitalized diabetic patients irrespective of their purpose of admission¹⁹. Every patient needs thorough evaluation and assessment for etiology & precipitating factors responsible for EI and therefore rational measures taken for their management.

Methodology

Study Design: It was a cross sectional study.

Study Subjects: The target population of this study was adult diabetic patients.

Place of the study: The study was conducted in the inpatients department of Medicine at BIHS & Hospital, Dhaka.

Study Period: This study was carried out for 8 months from January 2013 to August 2013.

Inclusion criteria:

- Adult (> 18 years) male and female patients.
- Suffering from DM.
- Admitted in the department of Medicine in BIHS & hospital.
- Identified to have electrolyte imbalance on admission.

Exclusion Criteria:

- Unwilling to be included in the study.
- Patient with advanced CKD (Stage 4 & Stage 5 CKD).
- Advanced CCF (New York Heart Association or NYHA Class 3 & 4).
- Persons with mental illness.
- Pregnant diabetic patient.

Sample size: This sample size was a total 150.

Sampling method: Sampling was made from all the admitted diabetic patients found to have electrolyte imbalance irrespective of their cause of admission and fulfilling the inclusion & exclusion criteria as mentioned. Data collection instrument: Semi-structured questionnaire and checklist.

Data collection technique: All participants were face to face interviewed using a semi-structured questionnaire. Anthropometric and clinical examination of each subject was recorded in the data collection sheet. Subjects were advised to give blood samples from the antecubital vein using disposable plastic syringe; 5 cc of blood was drawn and placed in test tubes for analysis of serum. Random blood glucose (RBG) on admission was done by bedside standardized glucometer from finger prick. Bedside readings by glucometer indicated "high" when RBG was > 30 mmol/L; which we have considered as severe hyperglycemia in this study. Urine for acetone bodies were done by collecting urine in a test tube and estimated by reagent strip method. The laboratory results were endorsed in the data collection sheet.

Data editing, coding, cleaning, and analysis: All the responses obtained from the patients were coded numerically and entered into the SPSS windows version 16.0 software program for analysis.

Results

The study was carried out among 150 diabetic patients admitted in the Medicine Department of BIHS & Hospital. Table 1 shows distribution of patients according to age, sex, education, occupation, residence and economic status of the total study sample of 150. Of the 150 subjects 7.4% belonged to the age group of 20 to 39 years, 45.3 % to the age group 40 to 59 years, and most (47.3%) belonged to the age group of 60 years and above. The lowest age was 25 years and the highest was 80 years. Their mean age was 56.8 years and standard deviation (SD) was + 12.5 years. Total number of male respondents was 56 (37.3%) and female, 94 (62.7%). Majority of the patients (73.3%) belonged to urban population. The rest belonged to semi-urban (15.3%) or rural area (11.4%). Financial condition was found to be of average in 52.7 %, good in 29.3 % and below average in 18.0 % of the study subjects. Most of the respondents (40.0%) had junior school certificate (JSC) level of education or less, followed by (34.6%) higher secondary school certificate (HSC), graduation and above level (14.0%) and no education at all (11.4%). Most were involved in household work (57.3%) followed by self employed (22.2%), doing government or private service (8.0%) and unemployed (12.7%).

 Table 1: Socio-demographic profile of the study samples (n=150)

Variables	Number	Percentage
Age(yrs)		
20-39	11	07.4
40 - 59	68	45.3
60 and above	71	47.3
Sex		
Male	56	37.3
Female	94	62.7
Education		
No formal schooling	17	11.4
Up to JSC (up to class VIII)	60	40.0
Up to HSC (up to class XII)	52	34.6
Graduation and above	21	14.0
Occupation		
Service (government/private)	21	08.0
Self - employed/business/agriculture	33	22.0
Household work/house wife	86	57.3
Unemployed/dependant	19	12.7
Financial Condition		
Average (13000 - 30000 taka/month)	79	52.7
Good (>30000 taka/month)	44	29.3
Below average (<13000 taka/month)	27	18.0
Residential Area		
Urban	110	73.3
Semi -urban	23	15.3
Rural	17	11.4

Figure 1: shows the distribution of type of diabetes. Type 2 diabetes (T2DM) was 94% and type 1, 6%.

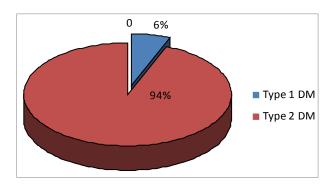


Figure 1: Type of diabetes (n=150)

Table-2 shows various treatments received by the patients till hospital admission as per their response. About 51.3% of study samples were on oral hypoglycemic agents and 37.3% on insulin therapy in addition to life style modifications (LSM) till hospital admission. Only a few (4.7%) of the diabetic study subjects said they did not receive any sort of treatment (neither drugs nor LSM).

Table 2: Treatment received till hospital admission(n=150)

Variables	Number	Percentage
Life style modification (LSM) only	01	00.7
Oral agents +LSM	77	51.3
Insulin +LSM	56	37.3
Oral drugs +Insulin +LSM	09	06.0
No treatment	07	04.7
Total	150	100

Figure 2, table 3 & table 4 shows diabetic and glycemic status of the study subjects at the time of hospital admission. Diabetes was found to be uncontrolled in most of the study patients (72%).

Ten percent of the subjects had severe hyperglycemia (RBG > 30 mmol/L) on hospital admission. Bedside readings by glucometer indicated "high" when RBG was > 30 mmol/L; which we have considered as severe hyperglycemia in this study as mentioned earlier. The mean \pm SD of RBG was 16.06 \pm 9.61, and the mean \pm SD of HbA1C was 9.91 \pm 2.79.

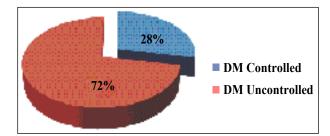


Figure 2: Status of diabetic control on presentation (n=150)

 Table 3: Patients having severe hyperglycemia on admission (n=150)

Variables	Number	Percentage
Severe hyperglycemia (RBG > 30 mmol/L)	15	10
Persons with RBG < 30 mmol/L	135	90

Table 4: Mean glycemic status at presentation

Variables	Mean ± SD
RBG	16.06±9.61
HbA1c	9.91±2.79

The pattern of electrolyte imbalance found in admitted diabetic patients irrespective of cause of admission has been shown in figure 3. Hyponatremia was found to be the commonest electrolyte imbalance (63.3%) among the subjects under study. The mean plasma sodium level of hyponatremic patients was 127.19 mmol/L (SD + 6.30). The lowest level of plasma sodium was found 109 mmol/L. Hypokalemia was found to be the second commonest pattern (46.7 %) of electrolyte imbalance. The lowest level of plasma potassium was 1.0 mmol/L. The mean plasma potassium level of hypokalemic patients was 2.83 mmol/L (SD+0.45). Hypomagnesemia was found to be the 3rd commonest pattern (8.7 %) and hyperkalemia being the fourth one (7.3%).

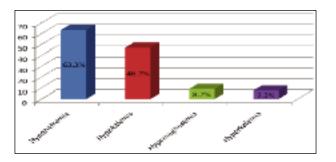


Figure 3: Patterns of Electrolyte Imbalance (n=150, multiple responses)

Table 5 shows the co-morbidities in the diabetic samples at the time of hospital admission. Sixty six percent subjects had Hypertension, 30% had stage 1-3 Diabetic CKD, 18.7% had Dyslipidemia, 15.3% had IHD, 4% had Cerebrovascular disease, 2.7% had CCF and 2.7% had Fatty liver.

 Table 5: Co-morbidities associated with Diabetes (n=150)

Co- morbidities	Number*	Percentage*
Hypertension	99	66.0
CKD (Stage 1-3)	45	30.0
Dyslipidemia	26	17.3
IHD	23	15.3
Cerebrovascular Disease	06	04.0
CCF (NYHA class 1 & 2)	04	02.7
Fatty Liver	04	02.7

*Multiple responses

Table 6 presents information on the precipitating factors of EI encountered in the admitted 150 diabetic patients under study. Vomiting was found to be the commonest (54.7%) precipitating factor for EI followed by diarrhea (23.3%). Other common precipitating factors for EI were severe hyperglycemia (10.0%), diabetic ketoacidosis (9.3%), pyrexia (10.7%), excessive sweating (7.3%), drug induced (11.3%), inadequate intake (10.7%), CKD (stage 1-3) (4%), CCF NYHA-class 1 & 2 (2%), and unknown (6.7%). Among the drugs found to cause EI, diuretics and ARB/ACE inhibitors were used for hypertension and CCF; whereas beta agonists and corticosteroids were used for bronchial asthma.

Table 6: Factors Precipitating Electrolyte Imbalance(n=150)

Precipitating factors	Number *	Percentage *
Vomiting	82	54.7
Diarrhea	35	23.3
Pyrexia (Fever)	16	10.7
Severe hyperglycemia	15	10.0
Diabetic Ketoacidosis	14	09.3
Excessive sweating	11	07.4
Drug induced	17	11.3
Inadequate intake	16	10.7
CKD (stage 1-3)	06	04.0
CCF (NYHA Class1 & 2)	03	02.0
No apparent cause detected	10	06.7

*Multiple responses

Total 95 patients were found to have hyponatremia among the 150 study subjects (63.3%). The factors precipitating hyponatremia revealed on hospital admission is shown in table-7. Vomiting was found the most common cause of hyponatremia (53.7%) followed by diarrhea (26.3%), severe hyperglycemia (12.6%), diabetic ketoacidosis (10.5%), pyrexia due to known or unknown cause (16.8%), excessive sweating due to pyrexia or other cause (11.6%), inadequate intake due to severe anorexia or voluntary salt restriction (11.6%), and drug induced (3.1%). No apparent cause of hyponatremia was found in 6.3% of case.

Precipitating factors	Number *	Percentage *
Vomiting	51	53.7
Diarrhea	25	26.3
Severe hyperglycemia	12	12.6
Diabetic Ketoacidosis	10	10.5
Pyrexia (Fever)	16	16.8
Excessive sweating	11	11.6
Inadequate intake	11	11.6
Drug induced	03	03.1
CCF	03	03.1
No apparent cause detected	03	06.3

Table 7: Factors Precipitating Hyponatremia (n=95)

*Multiple responses

Total 70 patients were found to have hypokalemia among the 150 study subjects (46.7%). The precipitating factors found responsible for hypokalemia has been shown in table 8. Vomiting was found responsible in 70% followed by diarrhea in 38.6%, inadequate intake in 11.4%, drug induced in 11.4%, and diabetic ketoacidosis in 7.1% of hypokalemic patients. No apparent cause of hypokalemia was found in 5.8% of case.

Table 8: Factors Precipitating Hypokalemia (n=70)

Precipitating factors	Number *	Percentage *
Vomiting	49	70.0
Diarrhea	27	38.6
Inadequate intake	08	11.4
Drug induced	08	11.4
Diabetic Ketoacidosis	05	07.1
No apparent cause detected	04	05.8

*Multiple responses

Total 13 patients were found to have hypomagnesemia (8.7%), and 11 hyperkalemia (7.3%) among the 150 study subjects. The factors responsible for hypomagnesemia and hyperkalemia have been shown in table⁹. The commonest cause of hypomagnesemia was found to be diarrhea (84.7%) followed by vomiting (69.2%), inadequate intake due to severe anorexia (7.7%) and unknown (15.4%). ARBs, ACEIs and K+ sparing diuretics like spironolactone, the drugs used for treatment of hypertension and other conditions was found to be responsible for 81.8 % of patients with hyperkalemia; the other one being CKD (54.5%). Diabetic ketoacidosis with severe hyperglycemia was found in 18.2 % cases of hyperkalemia in this study.

 Table 9: Factors causing Hypomagnesemia and Hyperkalemia

Variables	Number*	Percentage*
Hypomagnesemia (n=13)		
Diarrhea	11	84.7
Vomiting	09	69.2
Inadequate intake	01	07.7
Unknown	02	15.4
Hyperkalemia (n=11)		
Drug induced (ACEIs/ARBs)	09	81.8
CKD	06	54.5
Diabetic Ketoacidosis	02	18.2

*Multiple responses

Discussion

The main purpose of this study was to find out the type and the precipitating factors responsible for EI in admitted diabetic patients in an urban tertiary care hospital like BIHS & Hospital. In addition, the sociodemographic status and associated co-morbid conditions of those admitted patients were also evaluated. This cross sectional study showed that hyponatremia was the most common (63.3%) type of EI in admitted diabetic patients. It was found to be 80% in a study conducted in one of the largest hospital of Bangladesh mainly for diabetes care named Bangladesh Institute of Research & Rehabilitation in Diabetes, Endocrine and Metabolic Disorders (BIRDEM)¹⁹. Other studies have also shown that hyponatremia is common in hospitalized patients and is responsible for increased morbidity and mortality²⁰⁻²². Acute or symptomatic hyponatremia can lead to significant rates of morbidity and mortality²³⁻²⁵. In one study the mortality rate was $20\%^{26}$. Sterns reported a mortality rate of 5% when the serum sodium levels was $<105 \text{ mmol/L}^{27}$.

This study revealed that most of the patients were elderly, age being 60 years or more (47.3%). Hyponatremia is a common electrolyte disturbance in the hospitalized elderly sick patients²⁶. The incidence is much more in the elderly mainly owing to impaired ability to maintain water and electrolyte homeostasis in response to dietary and environmental changes¹⁸. In this study 62.7% of diabetic subjects having EI were female. Sixty percent of the cases in one prospective study on hyponatremia were female¹⁸. There was female preponderance of cases (55%) in another study²⁶. Studies have shown that female gender is an important risk factor for the development of severe complications²⁸. Chronic hyponatremia in postmenopausal women is common²⁹. Most of the patients in our study had multiple co-morbid conditions of which hypertension was the most common (66%). It was found to be 61%by a study conducted in BIRDEM¹⁹. Another study also revealed hypertension to be the most common (69%) comorbid condition in patients with hyponatremia²⁶. Vomiting was found to be the most common precipitating factor for EI in the admitted diabetic subjects of this study (54.7%). It was found to be 51 % by a study conducted in BIRDEM¹⁹. The next common cause of EI in our study was found to be diarrhea (23.3%). Drugs were responsible for 11.3% and severe hyperglycemia for 10% of cases of EI.

The most common precipitating factor for hyponatremia found in this study was vomiting (53.7%) followed by diarrhea (26.3%). Severe hyperglycemia associated with hyponatremia was found in 12.6% of study subjects. It is well known that hyperglycemia is associated with a decrease in serum Na+ concentration³⁰. Glucose being an osmotically active molecule, hyperglycemia can induce a fall in serum Na+ levels by shifting water from intra-cellular to extra-cellular compartments. It has been calculated that Na+ falls by 1.6 to 2.4 mmol/L for every 5 mmol/L rise in serum glucose levels³¹. Diuretics ARBs and ACEIs used for the treatment of hypertension or CCF can result in EI. In this study 3.1 % of patients developed receiving diuretics hyponatremia. Hyponatremic hypertensive syndrome is a well known entity, the most common association being in patients with essential hypertension receiving diuretics 32 .

Hypokalemia was found to be the 2nd commonest type (46.7%) of EI in this study. It has been stated to be one of the most common electrolyte disorder seen in both outpatient practice and inpatient care^{2,14,33}. Hypokalemia

is common in older adults, and increasing age is associated with a marked increase in the incidence of hypokalemia. In fact, in comparison to younger adults, an 80-year old has more than triple the risk of having a K+ level <3.0 mm/L^{2,14,33}. Gender also confers risk, with women more likely than men to have a low K+ leve^{12,14,33}. In one study, mortality of patients with hypokalemia was found more, particularly in females than those having normal K+ level³⁴. Hypokalemia is usually well tolerated in otherwise healthy people, but it can be life-threatening when severe¹⁴. Even mild or moderate hypokalemia increases the risks of morbidity and mortality in patients with cardiovascular disease. As a result, when hypokalemia is identified, the underlying cause should be sought and the disorder treated. Hypokalemia is almost always the result of K+ depletion induced by abnormal losses of potassium¹⁴. The most common precipitating factor for hypokalemia found in our study was vomiting (78%) followed by diarrhea (38.6%). Beta-2 agonist and corticosteroids used for bronchial asthma were found to cause hypokalemia in 11.4 % of our study. Diabetic ketoacidosis was found in 7.1 % cases of hypokalemia in this study. In uncontrolled DM, renal glucose loss causes osmotic diuresis, increasing Na+ delivery to the distal nephron and promoting K+ excretion. With prolonged glycosuria, there is considerable depletion of body stores of potassium⁴, but hypokalemia is usually mild or absent because both hypertonicity and insulin deficiency impede the entry of K+ into cells. The underlying K+ deficiency is rapidly unmasked when insulin is given, and severe hypokalemia can develop, particularly in patients with diabetic ketoacidosis, unless aggressive replacement of K+ stores is undertaken at the same time.

Hypomagnesemia was found to be the 3rd commonest type of EI in our study subjects (8.7%). And the precipitating factors encountered were diarrhea (84.7%), vomiting (69.2%), and inadequate intake due to severe anorexia (7.7%). Magnesium depletion induced either by dietary restriction or by abnormal loss, reduces the intracellular K+ concentration and causes renal K+ wasting³⁵. Magnesium deficiency should be corrected, particularly in refractory hypokalemia.

Hyperkalemia, also an important and sometimes a dangerous electrolyte disorder, was found in 7.3 % of the study subject. ARBs, ACEIs and K+ sparing diuretics like spironolactone, the drugs used for

treatment of hypertension and other conditions was found to be responsible for 81.8 % of patients with hyperkalemia; the other etiological factor being CKD (54.5%). Diabetic ketoacidosis with severe hyperglycemia was found in 18.2 % cases of hyperkalemia in this study. There is a study showing decrease in plasma Na+ and moderate increase in plasma K+ in diabetic patients with higher blood glucose concentration³⁶. In another study CKD, drugs and hyperglycemia precipitated hyperkalemia in 77%, 63% and 49% cases respectively³⁷.

Conclusion

Electrolyte imbalance was found to be very common in the elderly admitted patients of DM. Women were seen to develop EI more than men. Hypertension was the most common co-morbid condition associated with diabetes. Hyponatremia is an important electrolyte abnormality with the potential for significant morbidity and mortality. Because there are many causes of hyponatremia and the treatment differs according to the cause, a logical and efficient approach to the evaluation and management of patients with hyponatremia is imperative. Serum electrolytes should be measured in every admitted elderly diabetic patient irrespective of the cause of admission. The type of electrolyte imbalance and the precipitating factors responsible must be evaluated thoroughly and then managed accordingly. Drugs that cause electrolyte imbalance especially diuretics should be used with caution in the elderly. Among the other well established complications of diabetes like acute metabolic complications, microangiopathy and macroangiopathy, EI should also be considered especially in the admitted patients.

References

- Kerry CC. Electrolyte and Acid-Base Disorders. In: McPhee SJ, Papadakis MA, Rabow MW editors. Current Medical Diagnosis and Treatment. 51st ed. New York: Mc Graw Hill; 2012:pp.848-856.
- 2. Hawkins RC. Gender and age as risk factors for hypokalemia and hyperkalemia in a multiethnic Asian population. Clinica Chimica Act. 2003; 331:171-172.
- Frier BM, Fisher M. Diabetes Mellitus. In: Colledge NR, Walker BR, Ralston SH editors. Davidson's Principle and Practice of Medicine. 21st International ed. Churchill Livingstone Elsevier; 2010 :pp.798-814.
- 4. Atchley DW, Loeb RF, Richards DW Jr, Benedict EM, Driscoll ME. On diabetic acidosis: a detailed

study of electrolyte balances following the withdrawal and reestablishment of insulin therapy. J Clin Invest. 1933;12:297-326.

- Howard RL, Bichet DG, Shrier RW. Hypernatremic polyuric states. In: Seldin D, Giebisch G, editors. The kidney: physiology and pathophysiology. New York: Raven; 1991:p.1578.
- De Fronzo RA, Cooke CR, Andres R, Faloona GR, Davis PJ. The effect of insulin on renal handling of sodium, potassium, calcium and phosphate in man. J Clin Invest. 1975;55:845-55.
- 7. Zillich AJ, Garg J, Basu S, Bakris GL, Carter BL. Thiazide diuretics, potassium, and the development of diabetes: a quantitative review. Hypertension. 2006;48:219-24.
- Venkat Raman G, Albano JD, Millar JG, Lee HA. Bartter's syndrome and diabetes mellitus. J Intern Med. 1990;228: 525-31.
- Gorden P, Sherman BM, Simopoulos AP. Glucose intolerance with hypokalemia: an increased proportion of circulating proinsulin-like component. J Clin Endocrinol Metab. 1972;34: 235-40.
- 10. Janicic N, Verbalis JG. Evaluation and management of hypoosmolality in hospitalized patients. Endocrinol Metab Clin North Am. 2003;32:459-481.
- Schrier R. The patient with hyponatremia or hypernatremia. In: Schrier RW, editor. Manual of Nephrology. 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2000:pp21-36.
- Mount DB. Fluid and Electrolyte Disturbances. In : Long DL, Fauci A, Kasper D, Hauser SL, Jameson JL, Loscalzo J editors. Harrison's Principles of Internal Medicine. 18th ed. New York: McGraw-Hill; 2012:pp.344-359.
- Field MJ, Burnett L, Sullivan DR, Stewart P. Clinical Biochemistry and Metabolism. In: Colledge NR, Walker BR Ralston SH, editors. Davidson's Principle and Practice of Medicine. 21st International ed. Churchill Livingstone Elsevier; 2010: pp.435-446.
- 14. Gennari FJ. Hypokalemia. N Eng J Med. 1998; 339 :451 -458.
- Mandel AK. Hypokalemia & hyperkalemia. Med Clin N Amer. 1997;81(3):611-639.
- 16. Innerarity S. Hypomagnesemia in acute and chronic illness. Crit Care Nurs Q. 2000 Aug;23(2):1-19.

- Inzucchi SE, Bergenstal RM, Buse JB, Diamant M, Ferrannini E, Nauck M et al. Management of Hyperglycemia in Type 2 Diabetes: A Patient-Centered Approach. Position Statement of the American Diabetes Association (ADA) and the European Association for the Study of Diabetes (EASD). Diabetes Care. 2012 June; 35 (6) :1364-1379.
- Clayton JA, Le Jeune IR, Hall IP. Severe hyponatremia in medical in-patients: etiology, assessment and outcome. QJ Med. 2006;99:505-511.
- 19. Haque H, Amin MG, Uddin KN, Ahmed JU, Ahmed AKMS, Rahim MA et al. Pattern of electrolyte imbalance in hospitalized patients: experience in tertiary care hospital. Birdem Med J. 2012;2 (1):14-18.
- 20. Anderson RJ, Chung HM, Kluge R, Schrier RW. Hyponatremia: a prospective analysis of its epidemiology and the pathogenetic role of vasopressin. Ann Intern Med. 1995;102:164-68.
- 21. Chung HM, Kluge R, Schrier RW, Anderson RJ. Postoperative hyponatremia. A prospective study. Arch Inter Med. 1986;146:333-36.
- Areiff AI. Acid-base, electrolyte, and metabolic abnormalities. In: Parrillo JE, Dellinger RP, editor. Critical care Medicine: Principles of Diagnosis and Management in the Adult. 2. St. Louis: Mosby;2002.pp.1169-1203.
- 23. Arieff AI. Hyponatremia, convulsions, respiratory arrest, and permanent brain damage after elective surgery in healthy women. N Engl J Med. 1986;314:1529-35.
- 24. Arieff AI, Ayus JC, Fraser CL. Hyponatraemia and death or permanent brain damage in healthy children. BMJ. 1992;304:1218-22.
- 25. Ayus JC, Wheeler JM, Arieff AI. Postoperative hyponatremic encephalopathy in menstruant women. Ann Intern Med. 1992;117:891-897.
- 26. Rao MY, Sudhir U, Kumar TA, Saravanan S, Mahesh E, Punith K. Hospital-Based Descriptive Study of Symptomatic Hyponatremia in Elderly

Patients. JAPI. 2010 (Nov); 58:667-669.

- 27. Sterns, RH: Severe symptomatic hyponatremia: treatment and outcome. A study of 64 cases. Ann Intern Med. 1987;107:656-664.
- 28. Ayus JC, Arieff Al. Chronic hyponatremic encephalopathy in postmenopausal women: association of therapies with morbidity and mortality. JAMA. 1999;281:2299-2304.
- 29. Tareen N, Martins D, Nagami G, Levine B, Norris KC. Sodium Disorders in the Elderly. J NatI Med Assoc. 2005;97:217-224.
- Roscoe J M, Halperin M L, Rolleston F S, Goldstein M B. Hyperglycemia-induced hyponatremia: metabolic considerations in calculation of serum sodium depression. CMAJ. 1975 Feb;112: 452- 453.
- Hillier TA, Abbott RD, Barrett EJ: Hyponatremia: evaluating the correction factor for hyperglycemia. Am J Med. 1999;106:399-403.
- 32. Agarwal M, Lynn KL, Richards AM, Nicholls G. Hyponatremic-Hypertensive Syndrome with Renal Ischemia: An Under recognized Disorder. Hypertension. 1999;33;1020-1024.
- 33. Wenger NS, Roth CP, Shekelle P. Introduction to the assessing care of vulnerable elders-3 quality indicator measurement set. Journal of the American Geriatrics Society. 2007. 55: p. S247-S252.
- Lawson DH, Henry DA, Lowe JM, Gray JM, Morgan HG. Severe Hypokalemia in Hospitalized Patients. Arch Inter Med. 1979 Sep;139 (9):978-80.
- 35. Kobrin SM, Goldfarb S. Magnesium deficiency. Semin Nephrol. 1990;10:525-535.
- McNair P, Madsbad S, Christiansen C, Christensen MS, Transbøl I. Hyponatremia and hyperkalemia in relation to hyperglycemia in insulin-treated diabetic out-patients. Clin Chim Acta. 1982 Apr 8;120 (2):243-50.
- Acker CG, Johnson JP, Palevsky PM, Greenberg A. Hyperkalemia in Hospitalized Patients. Arch Intern Med. 1998; 158:917-24.