ORIGINAL RESEARCH PAPER
Volume - 14 | Issue - 02 | February - 2025 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

GASTROENTERITIS INDUCED POLYCYTHEMIA AND AKI : A CASE REPORT

Gastroentrology

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ABSTRACT

Summary: A 30 year old male patient presented with complaints of 18-20 episodes of loose watery stools and 10-12 episodes of vomiting for the last one day .He was admitted with the diagnosis of Acute Gastroenteritis with Severe Dehydration with Metabolic Acidosis . On examination he was found to have features of severe dehydration and on investigation he was found to have Polycythemia and AKI as well. His hemoglobin level was as high as 21.0 gm/dl which is very rare. On rehydration and other treatment all the three ailments got cured and the polycythemia which he had was Relative Polcythemia and not primary polycythemia .

KEYWORDS

Acute Kidney Injury (AKI) , Dehydration , Gastroenteritis , Polycythemia

INTRODUCTION

Gastroenteritis results in dehydration which may in turn, induce Polycythemia and Acute Kidney Injury (AKI).Multiple episodes of loose watery stools and multiple episodes of vomiting in our patient resulted in severe dehydration which induced Polycythemia and AKI.

In Polycythemia the values of hemoglobin , hematocrit or the red blood cell are elevated as compared to normal. Hemoglobin levels greater than 16.5 g/dl in women and greater than 18.5 g/dl in men and hematocrit value greater than 48 % in women and 52 % in men denotes polycythemia. Red blood cells production (erythropoiesis) occurs in the bone marrow where it is regulated in a series of specific steps. One of the important enzymes regulating this process is called erythropoietin (Epo). The majority of Epo is produced and released by the kidneys, and a smaller portion is released by the liver.

Polycythemia is of two types –Primary Polycythemia and Secondary Polycythemia. Primary Polycythemia is the result of internal problems with the production of red blood cells. In Secondary Polycythemia, polycythemia is caused due to another underlying medical problem. In primary polycythemia, overproduction of red blood cells occurs due to increased sensitivity or responsiveness to erythropoietin whereas in secondary polycythemia it results due to high levels of circulating erythropoietin which in turn may result due to chronic hypoxia or poor oxygen supply, an accompaniment to common conditions like lung or renal disorders. Patients suffering from polycythemia may be asymptomatic or present with symptoms pertaining to increased red cell mass or the underlying disease process responsible for increased red cell mass[1].

The hemoglobin values can also be raised due to reduction in the plasma volume as a result of dehydration and this type of Polycythemia is denoted as Relative Polycythemia [2]. In Relative Polycythemia cell volume is high due to increased blood concentration of red cells as a result of dehydration which in turn may be the outcome of vomiting, diarrhea or excessive sweating. Here the number of red blood cells is normal but blood plasma volume is relatively reduced because of the fluid loss and thus count of red blood cell seem to be elevated . Relative Polycythemia has variously been named as Chronic relative polycythemia or erythrocytosis or Pseudoerythrocytosis or pseudopolycythemia or apparent polycythemia. The last three terms are the most appropriate term for Relative Polycythemia. There is no polycythemia if there is no increase in red cell mass[3].

Dehydration affects the kidneys by forcing them to retain water and thus can result in Acute Kidney Injury which curtails the kidney function . Dehydration due to Acute Gastroenteritis can cause Acute Kidney Disease on account of hypoperfusion mechanism [4]. Haemoconcentration and increased plasma viscosity due to severe dehydration leads to Acute Kidney Disease and Myocardial Infarction [5]. Acute Gastroenterits can cause Acute Renal Insufficiency and likewise kidney ailment may cause gastrointestinal complaints. Seventy-five percent of patients with End Stage Renal Disease (ESRD) have gastrointestinal complaints. Hemodialysis plays an important role in improving the prognosis. [6]. The uremic retention products have substantial effects on cell and tissue function in many organ systems, including the gastrointestinal tract. The physiologic state of chronic uremia is likely to contribute to the development of the majority of symptoms. [7].

Case Report

A 30 year old male patient presented in the Medicine Department of SGT Medical college , Budhera , Gurugram with complaints of 18-20 episodes of loose watery stools and 10-12 episodes of vomiting for the last one day and had history of smoking 8-10 cigarettes a day. He was admitted with the diagnosis of Acute Gastroenteritis with Severe Dehydration with Metabolic Acidosis. On examination his tongue was found to be dry and eyes appeared to be slightly sunken. However , skin-pinch test was negative. He was found to have 110/70 mm Hg blood pressure , 84/mt. pulse , 18/mt. respiratory rate , 94% Spo2 and normal body temperature

Investigations On Admission :

C.B.C. investigations showed Haemoglobin as 21.0 gm/dl, R.B.C. as 5.8 /million/mm^3, were normocytic, normochromic with no NRBC or Target Cell and T.L.C. as 16700/cmm.

Kidney Function Tests showed Blood Urea as 54.0 mg/dl, Serum Creatinine as 03.67 mg/dl, Serum Uric Acid as 09.2 mg/dl, Calcium(Total) as 09.0 mg/dl, Serum Sodium as 128.0 mEq/L, Potassium as 3.2 mEq/L and Urine Exam. showed Traces of Albumin.

Liver Function Tests showed Serum Bilirubin Total as 2.1 mg/dl , Serum Bilirubin Direct as 0.3 mg/dl, Serum Bilirubin Indirect as 1.8 mg/dl, Serum SGPT as 39.0 U/L, Serum SGOT as 64.0 U/L, Serum Alkaline Phosphatase as 68.0 U/L , Total Proteins as 8.3 mg/dl, Serum Albumin as 4.3 mg/dl , Serum Globulin as 4.0 mg/dl and A G Ratio as 1.0 mg/dl

ABG Analysis (Morning / Evening) showed Ph as 7.288 / 7.265 , PCo2 as 27.0 mmHg/ 25.1 mmHg, PO2 as 86.0 mmHg/100.0 mmHg, HCo3 as 12.9 mmol/l / 11.4 mmol/l, TCo2 as 14.o mmol/l / 2.0 mmol/l and SPo2 as 96.0 % / 97 %.

Investigations On Discharge

C.B.C. investigations showed Haemoglobin as14.0 gm/dl, R.B.C. as 4.8 /million/mm^3, were normocytic, normochromic with no NRBC or Target Cell and T.L.C. as 6900/cmm.

Kidney Function Tests showed Blood Urea as 42.0 mg/dl, Serum Creatinine as 01.2 mg/dl, Serum Uric Acid as 06.3 mg/dl, Calcium (Total) as 09.4 mg/dl, Serum Sodium as 136.0 mEq/L, Serum Potassium as 3.5 mEq/L and Urine Exam. Showed no Albumin.

Liver Function Test Showed Serum Bilirubin Total as 1.0 mg/dl, Serum Bilirubin Direct as 0.2 mg/dl, Serum Bilirubin Indirect 0.4 mg/dl, Serum SGPT as 34.0 U/L, Serum SGOT as 39.0 U/L, Serum Alkaline Phosphatase as 61.0 U/L, Total Proteins as 8.4 mg/dl, Serum Albumin as 3.2 mg/dl, Serum Globulin as 3.1mg/dl and A G Ratio as 1.0 mg/dl.

ABG Analysis showed PCo2 as 36.0 mmHg, PO2 as 96.0 mmHg, HCo3 as 23.9 mmol/l, TCo2 as 25.o mmol/l and SPo2 as 97.0 % .

DISCUSSION

The patient presented with the chief complaints of loose watery stools

and episodes of vomiting for the last one day. He was, initially, diagnosed as a case of Acute Gastroenteritis. On detailed examination and investigations he was found to have Polycythemia, Renal Insufficiency and Metabolic Acidosis as well. This case was peculiar because of his hemoglobin level. His hemoglobin was as high as 21.0 gm/dl which is very rare.

He was initially treated for Gastroenteritis .When he started recovering from gastroenteritis, his CBC picture, kidney and liver profiles as well as his ABG picture also started improving. His Polycythemia vanished and kidney picture improved. His haemoglobin level came down to 14.0 gm/dl and R.B.C. count n to 4.8 /million/mm^3, Blood Urea to 45.0 mg/dl, Serum Creatinine to 01.2 mg/dl, Serum Uric Acid to 6.3mg/dl and albumin disappeared from the urine. Likewise his liver profiles and ABG picture also improved.

This shows that polycythemia in this case was not real but it was Relative Polycythemia which occurred as a result of severe dehydration caused by repeated episodes of loose watery stools and episodes of vomiting and it disappeared when dehydration improved. Acute Gastroenteritis, in the present case, besides causing polycythemia also caused. Acute Kidney insufficiency and both improved on recovery from Gastroenteritis. Lin WR et al reported one case of Acute Renal Failure from severe dehydration caused by S. enteritis food poisoning [8]. In our case Acute Gastroenteritis caused dehydration which in turn caused Relative Polycythemia and Acute Renal Injury which got recovered on rehydration.

Acute Gastroenterits induced Acute Renal Insufficiency is not uncommon in developing countries like ours. While treating a patient of Acute Gastroenterits, we must also look for the possibility of the presence Polycythemia and AKI. Early detection and referral if need be, of these patients will bring down mortality in these patients .

CONCLUSION

Whenever we come across any case of Acute Gastroenteritis , we must investigate and look for association of Relative Poyclythemia and / or Acute Kidney insufficiency. An early detection of Poyclythemia / AKI and early rehydration in a case of Gastroenteritis will avert the complications like chronic Kidney Disease.

REFERENCES

1. Christopher Haslett , Edwin R. Chilvers , John A.A. Hunter and Nicholas A.Boon: Davidson's Principles and Practice of Medicine18th Ed. 2001, 752 .
2. Jameson , Fauci , Kasper , Hauser , Longo and loscalzo. Harrison's Principles ofInternal Medicine Vol.2 , 20 th Ed.]
3. Lee: Wintrobe's Clinical Hematology, 10th ed.
4. Mohammed Hisham Bogari , Adeeb Munshi , Saleh Almuntashiri , Asim Bogari , Abdullah Shaker Abdullah , Mohammed Albadri , Ameer Hashim and Mohammed Saeed Alzahrani. Acute gastroenteritis related Acute Kidney Injury in a tertiary care center. Ann Saudi Med. 2023 , 6;43(2):82-89
5. Hosam Zaky , Sadeq Tabatabai , Parveez A Zarger and Jasem M AI Hashmi. A case Report of Severe Dehydration associated with Acute Kidney Injury causing Acute ST-Segment Elevation Myocardial Infarction. Cureus 2022; 22: 14(5) e25226
6. J Inbanthan and BU Lavanya. Clinical Profile of Renal Involvement in Acute Gastroenteritis Patients . Journal of Scientific Study. 2016, 4 (8): 48.
7. Babak Etemad . Gastrointestinal complications of renal failure . Gastrointestinal Diagnostic Laboratory , Philadelphia , 1998 ,Vol.27 (4) 875-89
8. Lin WR, et al. Diarrhoea associated acute renal failure in a patient with Salmonella enteritidis sepsis. Ren Fail. 2002.