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A CASE REPORT OF ATYPICAL HAEMOLYTIC UREMIC SYNDROME

Internal Medicine

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ABSTRACT

We report a case, 75-Year-old male, admitted in our tertiary care hospital with complaints of vomiting since 3 days and loose stool since 3 days. With initial evaluation, no specific diagnosis was made. After a hospital stay of 5 days and all lab reports a triad of MAHA(Microangiopathic haemolytic anaemia) thrombocytopenia, and AKI(Acute kidney injury) was satisfied and diagnosis of HUS was made. We managed the patient with fluids, antibiotics, and haemodialysis. Early diagnosis and treatment of patients with HUS can prevent patients landing up in End stage renal disease.

KEYWORDS

HUS, MAHA, AKI.

INTRODUCTION

Thrombotic Microangiopathy(TMA) is a primary disorder of renal microvasculature. 1]It is Usually associated with MAHA. When TMA has neurological manifestations it's called TTP and when it primarily involves kidney it is known as HUS. HUS is a triad of MAHA, thrombocytopenia and Renal impairment. We report a case 75-Year-old male, presented to our hospital with vomiting and loose stools and later diagnosed as HUS.

Case Report

A 75 year old male, farmer, presented with complaints of vomiting and loose stools since 3 days.He was apparently alright 3 days back when he developed vomiting which was associated with nausea, 2-3 episodes daily, watery not blood or bile stained.Also complains of watery loose stools 2-3 episodes per day, not blood stained or pus tinged.

Past history -

There was no h/o fever, Pain in abdomen , outside food consumption, cough/ cold / breathlessness . Patient has no past history of DM, HTN, TB, COPD, Epilepsy or immunocompromised state.No history of any addictions.No history of similar complaints in family members.

On clinical examination patient was conscious, alert, cooperative, well built. Patient had BMI of 24.4 and pulse rate of 134/min regularly regular, Low volume with no radio radial or radio femoral delay with all peripheral pulses palpable. Patient had blood pressure of 84/60 mm hg and spo2 of 97% on room air. Severe dehydration present .There was no pallor, icterus, cyanosis, clubbing, lymphadenopathy, edema.

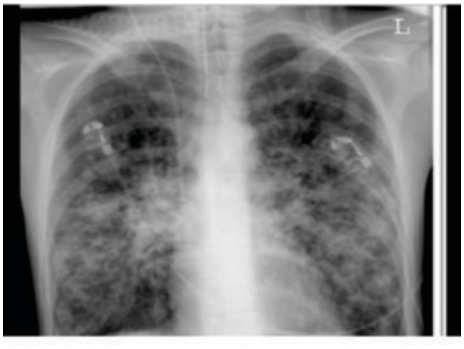

Image 1

On Systemic examination: In Cardiovascular examination both heart sounds were heard with no murmur. In respiratory system examination trachea shifted to left side and Breath sounds normal on both sides. In Neurological examination -No any neurological deficits, Per-abdomen examination: Abdomen is soft and non-tender. CNS examination: HMF normal, cranial nerves normal, sensory and motor system were normal

Laboratory investigations-

On admission day ECG WNL, CXR PA – WNL HIV, HBsAG, HCV ab – negative .Hb 11.2 , TLC 9200, Platelet 2.2 lakh, MCV 76,Peripheral smear – microcytic, hypochromic + RBC,S creat 1.78. BUL 62 RBSL 78. LFT details -bilirubin Total 0.6, direct 0.2, indirect 0.4, SGOT 27, SGPT 31, ALP 118 ,Protein Total 6.7, Albumin 4, Globular 2.7 ,PT 12 sec, INR 1.1 .Serum amylase 88, Lipase 98 ,Serum Na 141, k 3.8, Ca 10.1 mg/dl ,ESR- 18 mm/hr CRP 0.8 mg/LUSG a/p -R kidney – 10.6 × 4 cm ,L kidney – 10.7 × 3.9 cm, Both kidneys showing raised cortical echogenicity ,Bowel looks collapsed STOOL R/M shows on physical examination consistency watery,Mucus +,Blood + , parasites absent .

On Chemical examination Occult blood + Microscopic examination
WBC 8-10/hpf
RBC 12-15/hpf
Protozoans or cyst – absent
No organisms with darting motility seen.
Stool c/s at the end of 48 hours yielded no growth.

On 2nd day-

RFT- urea- 88, Creat-2.8

On 3rd day-

He developed breathlessness, ABG was s/o severe metabolic acidosis.
Cbc- Hb- 9.9, TLC- 9200, Plt- 68000
Urea- 157 creat- 4.1
Urine output - 400 ml over 24 hrs

On 4th and 5th day-

Hb, platelet count was reducing.
LDH- 908, indirect bilirubin -1.8, urine showed presence of Hb. and we repeated peripheral smear, it showed 2]schistocytes.

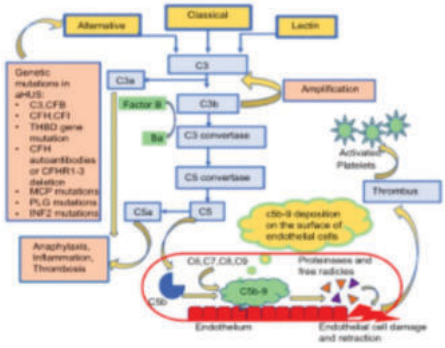

Image 2

On 6th day-

there was increase in level of indirect bilirubin and further fall in RFT and platelets. Based on clinical hisory and lab findings . Provisional diagnosis of HUS was made.From 7th day onwards patients showed improvement both Clinically and in lab parameters.

Treatment given- -

Initially CVP guided fluids ,Higher antibiotics ORS, Sporolac sachets given.After 24 Hours his CVP was increased , BP improved dehydration was reduced but output was low.After he developed Pulmonary edema secondary to AKI, we gave Lasix with caution.After diagnosis of HUS, we gave him blood transfusions and hemodyalysis was done.

Nephrologist opinion was taken and he advised to do 5 cycles of Hemodyalysis. Urine output was increased to 1.1 lt/ day.

Laboratory parameters were also improved with hb- 11.1, TLC- 6600, Plt- 2.6 lakh. Urea-54, creat- 1.6. After which patient was discharged and referred to Nephrologist.

DISCUSSION-

THROMBOTIC MICROANGIOPATHY (TMA)•3,4]TMA is a Pathological lesion characterized by endothelial cell injury in terminal arterioles and capillaries.

Platelet and hyaline thrombi causing partial or complete occlusion are histopathological features of TMA.

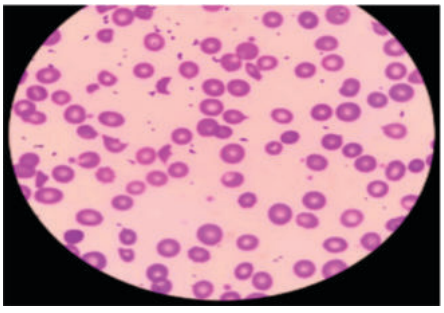

Image 3

TMA is usually accompanied by MAHA (microangiopathic hemolytic anemia) with its typical features of thrombocytopenia and schistocytes (but not always).TMA classically affect renal microvessels leading to progressive renal failure.

1. It is characterized by swollen endocapillary cells, fibrin thrombi, platelet plugs, arteriolar intimate fibrosis and membrano proliferative pattern in glomerulus.
2. Fibrin thrombi may extend into anterior vasculature pole,producing glomerular collapse and some times even cortical necrosis.
3. Those who recover from TMA, secondary FSGS may develop.

CONCLUSIONS

5]In cases Adult MAHA, high index of suspicion should be there for HUS. With prompt management, the complications like end stage renal disease can be prevented and morbidity and mortality can be reduced.

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