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PYELONEPHRITIS IN HEMOCHROMATOSIS PATIENT WITH CHRONIC LIVER DISEASE

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ABSTRACT

Haemochromatosis is an inherited disorder in which iron overload leads to increased chance of invasive bacterial infection. A 48 year old male patient was admitted in the medical ward with chief compliants of fever, abdominal pain, dysuria and backache. His medical history was Chronic liver disease with portal hypertension, varicoses, Antral and duodenal erosion, Diabetes mellitus. He was a mild alcoholic, stopped 1 year back. Elevated serum ferritin(800ng/ml), a-fetoprotein (180ng/ml). Urine culture shows the he avy growth of Ecoli (urine), staphylococcus aureus(sputum), CT evidence of focal lesion in liver, nephromegaly consistent with pyelonephritis, since iron overload may cause hepatotoxicity, hepatocarcinogenecity, and compromise the ability of phagocytes it should need to adjust dose(child pugh score=grade c), avoid iron supplements from own medications, proper antibiotic need to be provided to prevent the infection and phlebotomy has to be suggested to minimize the iron overload.

INTRODUCTION

Haemochromatosis is an inherited disorder in which excess iron overload can produce hepatocellularcarcinoma, cirrhosis, cardiac and gonadal dysfunction. Mostly the disease is seen in Northern Europe, with 1 out of 250 having this disorder where as in India the report on hereditary Haemochromatosis is rare.[7]

Over 93% of Hereditary Haemochromatosis are associated with mutations of two HFE genes namely C282y and H63D. About 75% of hereditary Haemochromatosis will be asymptomatic or may present with mild symptoms.[7] The high transferrin level increases the chance of invasive infection caused by bacteria. Escherichia coli has the ability to obtain iron siderophores and to secrete enterobactin in iron overload cases.[1]

Excess iron decreases chemotactic response against the infective organism and enhance the ability of invading organism to proliferate within the polymorph nuclear leukocytes by lowering its bactericidal capacity.[2] Formation of oxygen radicals by excess iron may produce toxicity to neutrophils, which alters the phagocytotic response of neutrophils. Patients with Haemochromatosis are at increased risk for heart diseases as the increased iron deposition in heart muscle may cause arrhythmia. Excessive iron depositions in pancreas may cause diabetes mellitus and pancreatic cancer.

CASE REPORT

A 48 year old male patient got admitted in the medical ward with chief complaints of fever, abdominal, pain, dysuria and backache. He had a medical history of Chronic liver disease with portal hypertension, varicose vein, Antral & duodenal erosion and diabetes mellitus. His social history showed that he was an alcoholic and has been a teetotaler since one year. His medication history included folic acid, Propranolol, Telmisartan and Insulin. On examination the patient was febrile (39.2ºC), conscious, oriented, and his other vitals were found to be normal. He had a bronzed complexion with scleral icterus and palmar erythema. His abdominal examination revealed hepatomegaly with moderate ascites.

Laboratory examination revealed the presence of thrombocytopenia with elevated levels of total count (16000 cell/ mm), random blood sugar, international normalized ratio and slight hyponatremia. Liver function test showed marked abnormality with total bilirubin of 5.2mg/dl, elevated level of alanine transferase, aspartate aminotransferase, hypoalbunemia(<2.6mg/dl). Elevated levels of serum ferritin (800ůg/ml) and α-fetoprotein (180ng/ml) revealed the presence of hepatocellular carcinoma. After the initiation of drug therapy serum creatinine was elevated form 0.8mg/dl to 1.6 mg/dl. CT of abdomen showed focal lesion in liver, ascites, features suggestive of chronic liver disease, portal hypertension and nephromegaly consistent with pyelonephritis. Urine culture had heavy growth of E coli and sputum culture revealed the presence of staphylococcus aureus. Patient experienced bronzed complexation over the extremities due to existing iron overload.

Treatment Given

Since the patient had pylonephrities, the main goal of therapy was to eliminate the infection. Antibiotic therapy with a combination of broad spectrum antibiotics was initiated to attain the same. Treatment was iniated with Inj Gentamicin 160mg once daily followed by Inj.Levofloxacin 500mg once daily, Tab Cefixime 200mg once daily and Piperazilin

tazobactum 4.5gm Q8H. Portal hypertension was treated with Tab propranolol 20mg twice daily. Supportive therapy for symptomatic relief was also provided.

Based on urine culture sensitivity, Gentamicin was initiated and was later discontinued as the patient experienced drug induced nephrotoxicity. Later levofloxacin was introduced and was discontinued 4 days later as the patient was febrile and had cutaneous rashes. Patient was then given cefixime but due to the occurrence of thrombocytopenia, it was discontinued. This was followed by the use of piperacillin tazobactum.

DISCUSSION

Serum iron level is increased in Haemochromatosis. This is accompanied compromised host defense mechanism that leads to increased chance for various bacterial and non bacterial infections. The excess iron not only helps the propagation of pathogen, but also plays an important role in modifying the host immune response. Impairment of Hepcidin due to iron overload may lead to increased susceptibility for infection in the patients. This patient had a *gastrointestinal discomfort, hepatic imp*airment, was febrile and had heavy bacteremia in urine and sputum. He also had elevated levels of serum ferritin (800ůg/ml) and α-fetoprotein (180ng/ml) which were suggestive of hepatocellular carcinoma.

During treatment effective antimicrobials like inj Gentamicin, cefixime, levofloxacin and piperacillin tazobactum was used to treat the infections due to iron overload but drug dosing was not based on child Pugh score (class c). This lead acute renal failure and thrombocytopenia. The patient is already on severe iron overload and further intake of folic acid and cynocobalamin containing vitamin tablets may lead to increased iron content in the blood which may again worsen the patient condition. Untreated heamochromatosis can lead to cirrhosis, endocrinopathies, cardiomyopathy, skin pigmentation, Hepatocellular carcinoma which may reduce life expectancy. Here the patient requires proper antimicrobial therapy to avoid septic shock as a result of iron overload.[4] Other study shows that prognosis of untreated Haemochromatosis is poor, where early diagnosis and phlebotomy can improve the survival rate.[5]

CONCLUSION

In this case the patient diagnosis with hemochromatosis is an incidental finding and clinician should need to consider various pathogens in patient due to iron overload, modulation of iron metabolism by using recombinant hepcidin. Should need to adjust dose (childpugh

score=grade c), avoid iron supplements from own medications, proper antibiotic need to be provided to prevent the infection and phlebotomy has to be suggested to minimize the iron overload.

Other consideration that should be considered in the field of hemochromatosis is to develop new markers for genetic screening. It will be highly effective to screen the population with the genetically predisposed risk to get screened in the initial life stage.

The treatment plan in case of these patients has to be tapered based on the child pug score. Thus the drug induced toxicities can be reduced to higher extent in the clinical settings.

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