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CASE REPORT: ATYPICAL PRESENTATION OF ENTERIC FEVER

Internal Medicine

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ABSTRACT

Enteric fever is an invasive bacterial infection acquired through consumption of contaminated water or food. The organism classically responsible is *Salmonella enterica* serotypes Typhi and Paratyphi A, B and C. Enteric fever usually presents with abdominal pain, fever, and chills approximately 5 to 21 days after ingestion of causative microorganism.[1] Classic manifestations include relative bradycardia, pulse-temperature dissociation and "rose-spots". Laboratory findings may include anemia, leukopenia, leukocytosis, and abnormal liver function tests.Atypical presentations include Abdominal Lymphadenopathy, Acute acalculous cholecystitis, Jaundice, Pancreatitis, Meningitis, Parotitis, Orchitis etc.[2] We herein report a rare atypical case of enteric fever which presented with fever, thrombocytopenia and hyperbilirubinemia.
With changing patterns of typhoid fever timely diagnosis is necessary to prevent complications such as peritonitis due to perforation. Typhoid hepatitis is also being considered as a different entity, with increasing yet rare cases.

KEYWORDS

Enteric-fever, Widal, Hepatitis, Thrombocytopenia , Antibiotics

CASE DESCRIPTION:

A 26 year old male presented with the chief complaint of fever for the last 7 days and loss of appetite for the last 2 days. Patient developed petechial spots all over the body. No stomach ache, diarrhea, nausea, vomiting.

Findings are as follows: temp-102F, icterus present, mild hepatosplenomegaly on examination.

Lab: Hb – 13.2gm%, TLC 3900/cumm (N65, L30, M3, E2), Platelets: 60,000/cumm, ESR: 18, Total Bilirubin 7.8mg% (direct: 6.6), ALT 239, AST199, ALP 232.PBF: normocytic normochromic picture with thrombocytopenia.

Serum electrolytes, aPTT/PT and RFT normal.
Dengue IgM and leptospira IgM were negative
Usg abdomen: hepatosplenomegaly.
Widal test: 1:320, highly positive
Blood cultures grew Salmonella after 2 days of incubation.

Patient developed petechial spots that worsened on day 2 of admission with platelet count dropping to 28,000/cumm. He was treated with parenteral ceftriaxone, ofloxacin and IV fluids. The patient became afebrile after 3 days, jaundice started improving and petechial rash disappeared. Total bilirubin came down to 2.4mg/dl on the 7th day of admission. Platelets increased to 92,000/cumm. He was discharged after 10 days on oral cefixime and ofloxacin.

DISCUSSION :

Enteric fever should be suspected in a febrile patient living in endemic area, particularly if the duration of fever is more than 3 days without alternative explanation. Our patient with typhoid fever had thrombocytopenia and hepatitis as presenting symptoms, without any other classic signs like stomach ache or constipation etc. Proposed mechanisms of thrombocytopenia in typhoid fever include bone marrow suppression, peripheral destruction by reticuloendothelial system, autoimmune-induced destruction and *Salmonella* endotoxin-induced thrombocytopenia. The pathogenesis of hepatic involvement in typhoid can either be due to direct invasion by salmonella or else the endotoxin damages the hepatocytes with reticulo-endothelial cell hyperplasia.

Very important to note that all jaundice and hepatitis are not viral, as is proven by this case.

Since the risk of hemorrhage increases exponentially with the fall in platelet count , luckily our patient didn't go below the 20,000/cumm threshold mark for platelet transfusion. The thrombocytopenia and hepatitis both responded with conservative management and antibiotics, which were started on time with timely diagnosis.

CONCLUSION:

We have described a case of severe thrombocytopenia and hyperbilirubinemia in typhoid fever with slow clinical and laboratory response but complete recovery after appropriate antibiotic therapy and supportive care alone, thus avoiding the complications related to platelet transfusion and plasmapheresis. Clinical presentations of typhoid fever vary from case to case.

Fever along with jaundice and thrombocytopenia is usually seen in malaria, dengue and leptospirosis. However, differential diagnosis of typhoid fever must be kept in mind in a febrile patient with jaundice.

This case report is aimed to sensitize the physicians that typhoid infection can present with fever, frank jaundice and thrombocytopenia. The optimal management of rare manifestations typhoid fever merits further study to guide clinical practice.

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