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Research Article

IS OVERWHELMING SYSTEMIC SEPSIS AN UNANTICIPATED CONSEQUENCE OF IRON THERAP ?– A RARE CASE REPORT

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ABSTRACT

Iron deficiency is the most common nutritional deficiency world wide. The World Health Organisation recommends universal iron supplementation of pregnant women and young children in developing countries. It is a well known fact that treatment with iron supplements is associated with gastrointestinal adverse effects such as constipation, nausea, metallic taste, diarrhoea, epigastric distress and vomiting as well as the occasional severe anaphylactic reactions. In rare instances, therapeutic treatment with iron may be associated with increased risk of infection as bacteria and other infectious agents also utilize iron as a growth factor. Hereby, we report a rare entity known as sepsis syndrome which surfaced in a 20year old female after receiving parentral iron therapy.

INTRODUCTION

Iron Deficiency is one of the prevalent forms of malnutrition. The prevalence of ID in mothers and young children frequently exceeds 50% in low-income countries[1,2]. World Health Organisation recommends universal iron supplementation of pregnant women and young children[3].

The most common side effects associated with iron therapy are constipation, dark stools, stomach pain, nausea, and vomiting. However, Studies suggest that iron supplementation also increases susceptibility to bacterial infections and increases the virulence of many common bacterial enteropathogens[4-5]. Iron lies at the centre of a battle for nutritional resource between higher organisms and their microbial pathogens.The ability of the host to reduce the availability of free ionic iron to invading pathogen is one of the most evolutionarily conserved innate strategies against infection[6]. To successfully sustain an infection, nearly all bacteria, fungi and protozoa require a continuous supply of host iron. Most bacterial species have evolved counter-acting strategies to avail the iron through receptors that bind transferrin, lactoferrin or haemoglobin; and low molecular weight siderophores that acquire iron from host proteins or from low molecular weight iron compounds. Increased rates of malaria, respiratory infections, severe diarrhoea and febrile illnesses of unknown origin have all been reported .We report one case from Bhubaneswar , Odisha where the patient developed sepsis syndrome after receiving Iron therapy.

Case Report - 20yr old Hindu female was admitted to Apollo hospital , Bhubaneswar with c/o fever , loose motion , vomiting – multiple episodes and headache for 1 day . The patient had history of menhorrhagia , iron deficiency anaemia with no other comorbidities and had haemoglobin level – 6. 7 gm% . She had received 3 Parentral Iron injections as advised by gynaecologist within past 7 days in another hospital and had consumed outside food one day prior to developing symptoms. On admission, the patient was febrile – 103 ^{o}f , Tachycardic – 130/ min, tachypnoec, Blood pressure – 70/ 50 mmhg . On General examination - Patient had blanchable rashes on her body and had pallor. Systemic examination was not significant. Arterial blood gas analysis showed raised lactate level with metabolic acidosis. Patient was resuscitated with isotonic IV fluid bolus. Blood pressure did not improve. Patient was started on Vasopressor support to maintain MAP > 65mmhg.

Lab investigation showed HB – 8.6 gm % , TLC – 8670 / cm ^{3}, Platelet count – 200000/ cm ^{3} , ESR – 60mm/ 1st hr , LFT was deranged s. total bilirubin – 3.5mg/dl, Direct bilirubin – 2.4mg/dl, indirect bilirubin – 1.1 mg/dl. AST – 62 u/l , ALT -, ALP – 36, albumin – 2.8gm/dl . Renal function tests deranged

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Creat – 1.75 mg/dl , urea – 63mg/dl . Electrolytes Na + - 124 , K + - 3.5 , Mg – 1.2 , Calcium – 7.5. Coagulation Profile deranged PT -26. 5, APTT - 44.7, INR – 2.35 . Raised inflammatory marker CRP (Q) – 116 .Serum Procalcitonin – 47 ng/ml s/o sepsis. USG Whole abdomen showed mild hepatosplenomegaly, minimal POD fluid. Chest X ray was within normal limits .Malaria(ICT) – negative , Dengue (IgM , IgG , Ns1ag) – negative , Leptospira (Igm) & Scrub typhus (Igm) was negative .Viral Serology were negative. Urine showed presence of 6-7 pus cells. Patient fluid intake was restricted to 1.5l / day. Patient was started on Broad spectrum antibiotics (carbapenems & teicoplanin) in renal modification dose with other supportive measures.

On 2nd day of admission , Patient Urine output improved and patient was off vasopressor support. On 3rd day blood C/S showed presence of Staphylococcus pseudointermedius which was resistant to Fluroquinolones, macrolides but sensitive to linezolid, teicoplanin, Vancomycin and carbapenems . Patient was already on those antibiotics which was sensitive . Patient improved symptomaticaly . On 5th day Repeat LFT ,RFT , Pt/INR was done . Creatinine – 0.56 , urea – 10 , t. bilirubin – 0.9 , Direct. bilirubin – 0.2 , AST – 35 , ALT – 47 , ALP – 46 .PT – 18.2, INR – 1.42 . All the parameters had improved after administration of proper Antibiotics. Patient improved symptomatically and was discharged in a clinically stable condition afterher 6thday of hospitalization.

She was diagnosed as a case of SEPSIS SYNDROME
(Staphylococcus Pseudointermedius)

DISCUSSION

Iron is a critical nutrient for both humans and pathogenic bacteria. In humans, iron-containing compounds participate in a number of important cellular processes including energy metabolism, cellular proliferation, DNA repair, and protection against oxidative stress. However, iron is also essential to invading bacterial pathogens; therefore vertebrates sequester iron as an innate immune response against bacterial infection. Staphylococcal species is a pathogenic bacterium that requires iron to carry out vital metabolic functions and cause disease. The most abundant reservoir of iron is present inside the heme. Utilization of exogenous heme by bacteria involves the binding of heme or hemoproteins to the cell surface receptors, followed by the transport of heme into cells. Once taken into the cytosol, heme is presented to heme oxygenases where the tetrapyrrole ring is cleaved in order to release the iron[7-9].

Murray *et al*[10] stated that the incidence of infections in iron deficient somali nomads who received iron therapy was higher than the group who received placebo.

Canziani *et al*[11] found that the risk of infection was higher with higher intravenous doses of iron than with lower doses.Collins *et al*[12] found a higher risk of sepsis and hospitalization in patients who received iron for a prolonged duration (5–6 months) than in those who did not.

Barry D M *et al*[13] that there is increased incidence of gram-negative neonatal sepsis with iron administration based on the disastrous outcomes of intramuscular iron-dextran administration to Polynesian neonates.
Jaeggi.T *et al*[14] stated that Iron fortification adversely affects the gut microbiome, increases pathogen abundance and induces intestinal inflammation in Kenyan infants as iron is essential for growth and virulence of many pathogenic enterobacteria.

Esan *et al*[15] stated that Iron supplementation in anemic HIV-infected children has beneficial effects on hemoglobin, anemia, and immunity but increases the risk of malaria. Thus, iron supplementation in HIV-infected children living in malaria-endemic areas should only be provided in combination with adequate protection from malaria.

CONCLUSION

The patient who was previously asymptomatic and had no co-morbidities develops septic shock after receving iron therapy . The exact role of iron in escalating the infection needs to be determined as it lies at the epic centre of the host – pathogen battle for resource control . The policy of mass administration of iron therapy in developing countries should be limited to minimise the hazardous effect of potentiating pathogen resulting in septicaemias .Prompt management by the treating physician is required in case of septicaemia .So, The importance of this article lies in the awareness of the existence of such entity so that prompt diagnosis and timely treatment can be instituted.

Prior Publication

This article has not been published or submitted for publication elsewhere, in whole or in part, before submission to the Case Reports in Critical Care.

Consent

The authors declare that they have provided written informed consent from the described patient for the case report to be published. Conflict of Interests: The authors declare that there is no conflict of interests regarding the publication of this paper.

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