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Case Report

Culture proven Salmonella typhi co-infection in a child with Dengue fever: a case report

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Abstract

Infectious diseases are one of the major causes of morbidity and mortality in developing countries. Sometimes concurrent infections with multiple infectious agents may occur in one patient, which make the diagnosis and management a challenging task. The authors here present a case of co-infection of typhoid fever with dengue fever in a ten-year-old child and discuss the pertinent issues. The authors emphasize that the risk factors predicting the presence of such co-infections, if developed, will be immensely useful in areas where dengue outbreak occurs in the background of high transmission of endemic infections.

Key words: enteric fever; dengue; co-infection; child.

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Introduction

In developing countries, infectious diseases contribute considerably to morbidity and mortality. Sometimes concurrent infections with multiple infectious agents may occur, which make the correct diagnosis and management a challenging task [1]. The authors here present a case of co-infection of typhoid fever with dengue fever in a child and discuss the pertinent issues. Clinicians should be aware of the potential for concurrent bacteremia when treating patients with dengue infection.

Case Report

A ten-year-old male child presented to the pediatric emergency with complaints of high fever for six days and associated upper abdominal pain for four days. The fever was of a continuous nature and was not associated with chills and rigors. He also complained of non-projectile vomiting. Four days prior to presentation, the child was noticed to have facial swelling and abdominal distension. Parents did not observe any rashes or spontaneous bleeding.

On examination, he had facial puffiness and multiple petechiae over his chest. He was febrile with an axillary temperature of 102˚F. His pulse volume and blood pressure were normal. Abdominal examination revealed a distended abdomen with flank fullness. Liver was tender and palpable four cm below right costal margin and spleen was felt one cm below left costal margin. Air entry was reduced in the right infra-scapular and infra-axillary regions with a dull note being elicited on percussion. Examination of the cardiovascular and central nervous systems was normal. There was no evidence of any cutaneous eschar.

In view of the typical presentation of fever with clinical evidence of capillary leak and petechiae, a diagnosis of dengue fever was considered at admission. His packed cell volume (PCV) at presentation was 40 and platelet count was 21000/cu.mm. Peripheral smear revealed leukopenia with neutrophilic toxic change (4200/cu.mm, Neutrophils 85%). Chest X-ray showed bilateral pleural effusion. Ultrasound abdomen confirmed the presence of hepatosplenomegaly with ascites. Liver and renal function tests were normal (Bilirubin- 0.4 mg/dL, Aspartate transaminase- 50 IU/L, Alanine transaminase- 44 IU/L, urea- 24 mg/dl, creatinine- 0.6 mg/dl). ELISA tests for NS1 antigen and IgM antibody for dengue were positive, confirming the diagnosis of dengue.

The child was started on an intravenous infusion of ringer lactate at 5 ml/kg/hr. Serial monitoring revealed normal urine output and falling trend of hematocrit after intravenous fluids. The infusion rate was gradually decreased and stopped by the second day of

hospital admission. By day five of admission his hematocrit on oral feeds was 30 and his platelet count had risen to 40000/cu.mm. The peripheral blood smear showed leukopenia with neutrophilic toxic change (4400, Neutrophils 73%). However, in spite of apparent clinical improvement, the fever spikes persisted. In view of persisting fever, blood samples were sent for serology and culture considering the possibility of scrub typhus and typhoid fever. Widal test was strongly positive, titres against O antigen was 1:160 and H antigen was 1:640. Weil Felix test was not suggestive of Rickettsial illness. Based on the Widal test result, the child was started on intravenous ceftriaxone. Subsequently, the blood culture also yielded *Salmonella typhi* sensitive to ceftriaxone, ampicillin and ciprofloxacin. After five days of treatment, the fever subsided and he was discharged after receiving 10 days of ceftriaxone.

Discussion

Our patient most probably had co-infection of dengue and typhoid. Typhoid fever is endemic in India and it is most prevalent in urban areas, with incidence approaching one percent of the population annually in some endemic areas [2]. In the age group between 5 and 15 years the reported incidence of typhoid fever is 214.2 per 100,000/year [3]. In India, dengue virus causes epidemic and sporadic cases year-round, with a peak in frequency from August to November, during the humid season [4]. Co-infections with common endemic pathogens can prove to be a diagnostic challenge especially during dengue outbreaks.

Many co-infections with dengue have been reported from tropical countries including malaria [5], melioidosis [6] and chikungunya [7,8]. Dual infections with other endemic diseases, such as leptospirosis, viral hepatitis B also have been reported in cases with unusual manifestations [9].

Though co-infections of dengue and typhoid have been reported in few adult patients [10,11], there are very few reports in pediatric patients to date. Co-infections of dengue and typhoid have been observed in large case series of febrile children [12,13]. Our patient initially tested positive for NS1 antigen and also for IgM antibody for dengue suggesting an acute infection with dengue. The initial presentation of fever with clinical and radiological evidence of capillary leak, along with a fall in hematocrit of > 20 % with intravenous fluids, was compatible with the diagnosis of dengue. However, the child had persistent fever despite signs of recovery from dengue suggested by resolution of clinical fluid accumulation and improvement in platelet count. This prompted us to evaluate him for other co-infections. Widal test was strongly positive and *Salmonella typhi* was isolated in blood culture.

Risk factors for bacterial co-infection in children with dengue have not been well characterized. In a study done in adult patients, prolonged fever (> 5 days) was an independent risk factor for co-infection [14]. The reasons for bacterial co-infections in some patients with dengue are also not yet fully known. It is known that dengue virus can cause a diminished T cell proliferation in response to mitogens *in vitro* [15]. However, the *in vivo* effects of these observations have not been studied. With increasing reports of co-infections in patients with dengue, it would be worthwhile to study the immune effects of dengue *in vivo* (*eg*. delayed type hypersensitivity to purified protein derivative). Another probable reason cited for increased gram negative sepsis with dengue is the breakdown of intestinal mucosal barrier [14].

Attempts have been made to identify risk factors predicting presence of bacterial co-infection in adult patients with dengue [16], but similar studies have to be done in pediatric age group.

Conclusion

Our patient had a co-infection of dengue and typhoid and this highlights the importance of considering a co-infection in children being treated for dengue fever in the presence of an atypical clinical course. The risk factors predicting the presence of such co-infections in pediatric age group, if developed, will be immensely useful in areas where dengue outbreaks occur in the background of high transmission of endemic infections.

Authors' Contributions

Rangan Srinivasaraghavan, Kanimozhi T and Narayanan Parameshwaran managed the patient. Rangan Srinivasaraghavan and Narayanan Parameshwaran reviewed the literature. All authors contributed to drafting of the manuscript and approved the final version of the manuscript.

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