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CASE REPORT

YOUNG BOY HAVING ENTERIC FEVER PASSED THROUGH ICU DUE TO ACUTE RESPIRATORY DISTRESS SYNDROME LUCKILY SURVIVED

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Abstract:

Being part of a developing world, most of our population suffers from various infectious diseases, enteric fever is one of the commonest of all. A greater percentage of affected children and young adults recover without any complications. As its initial symptoms are very benign, sometimes proper treatment is delayed and that can lead to morbid situation. Our presented case is important to get reported because here a young man was about to die due to one of the rarest respiratory complications of enteric fever that very rapidly led to MODS. Fortunately, he survived for all the prompt clinical judgements were taken in time.

Key words: *Enteric Fever, ICU, Acute respiratory distress syndrome (ARDS)*

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Introduction:

Enteric fever, a systemic infection caused by *Salmonella enteric* serovar Typhi (*S. typhi*) and Paratyphi (*S. paratyphi*), is a common cause of morbidity and mortality in the developing world, particularly in South and South East Asia. Highest incidence has been documented in impoverished, overcrowded areas where poor sanitation infrastructure facilitates faeco-oral transmission[1]. More than 27 million cases with 2,16,000 deaths of enteric feveroccur annually worldwide with the majority of disease burden being borne by children and adolescents[2]. Children usually presents with highgrade fever, coated tongue, nausea/vomiting, diarrhea, constipation, hepatomegaly, splenomegaly, abdominal distension, GI bleeding, anemia, and neutrophilia. In adults' predominantly nausea/ vomiting, thrombocytopenia and GI perforation.Among culture–confirmed ambulatory enteric cases hospitalization rate is up to 2–40%[1]. Acute respiratory distress syndrome (ARDS) is a very rare complication of enteric fever and has been recorded only a handful of times. Here, we are presenting the case of a young man suffering from enteric fever battled with this grave complication.

Case report:

This 21-year-old university student initially presented with gradually increasing high grade fever, consulted with a local physician, started taking oral Azithromycin, but didn't improve. Within the first week, fever followed a step-ladder pattern and gradually increasing weakness pursued. On routine check-up, CBC showed severe anemia and blood culture report suggested growth of Salmonella typhi, which was sensitive to all antibiotics. Afterwards, he

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got admitted to a hospital at Gazipur and treatment started with I/V Ceftriaxone 2g twice daily and he received 2units of whole blood for severe anemia. On query he replied that he had black soft stool for two days.

Despite appropriate treatment, he was kept getting worse. His febrile condition was unchanged with abdominal pain, and melena. Furthermore, he developed acute onset of severe respiratory distress on 10th day of his illness. SpO_2 lowered to 70% in room air; needed 10L O_2/min via face mask initially and then switched to NRBM to maintain SpO_2 above 95%. When SpO_2 kept falling despite all these, he got transferred to a tertiary care hospital in Dhaka for ICU care.

AtPMCH ICU, his vitals showed temperature of 104.5°F, pulse 118/bpm, BP 140/80 mmHg, RR 37/min; was severely anemic, mildly icteric and edematous with just palpable spleen. SpO_2- 93% with 15LO_2/min via NRBM, andon auscultation of chest-bilateral coarse crepitations were found. Immediate portable chest x-ray was done [Figure-1].

He had been primarily diagnosed to have ARDS, based on having culture positive enteric fever as the precipitating illness, hypoxemia and typical chest x-ray finding. PCWP was not measured. I/V Corticosteroid, Frusemide and Ciprofloxacin were added along with ongoing Ceftriaxone. His oxygenation needed to be maintained with high flow nasal cannula a with the maximum flow rate of 60L/min.

His blood reports on admission showed severe normocytic normochromic anemia, with slightly raised reticulocyte count, S. bilirubin, LDH; altered LFT, hypoalbuminemia and high level ofacute phase reactants.

Both direct and indirect Coomb's test and ANA was negative that ruled out hemolysis or any autoimmune cause for severe anemia. Stool OBT was positive. He

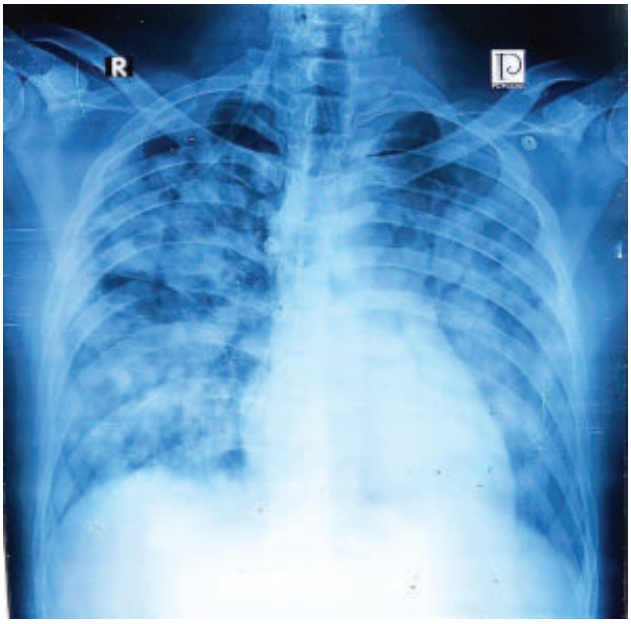

Fig.-1: *Portable chest x-ray showing bilateral extensive pulmonary infiltrates.*

received another unit of whole blood transfusion in ICU. Ultrasonogram of whole abdomen suggested mild hepatosplenomegaly, bilateral pleural effusion and ascites.ECG and Echocardiography were normal. Sputum and throat swab were also sent for culture but showed no growth.

On 12th day of illness, he became semiconscious (GCS 11/15),no focal neurologic deficit was present but started talking incoherently regarding the fear of death, which responded well with I/V Haloperidol. At this state, he finally diagnosed as a case of "Complicated multi-drug resistant enteric fever with MODS". His total antibiotic regime had been switched to I/V Meropenem 2g TDS, and I/V Teicoplanin.

Within the next 5days with this treatment protocol, he got improved enough to be transferred to cabin from ICU being fully conscious, afebrile and SpO_2 maintained in room air.

Hb (g/dl)	Platelet (/cmm)	TC WBC (/cmm)	ESR (mm/hr)	SGPT (U/L)	SGOT (U/L)	S. Bilirubin (mg/dl)	S. Albumin (g/dl)
4.90	295,000	9.26k	67	105	83	T- 1.87 I- 1.25 D- 0.62	2.78

Reticulocyte count (%)	CRP (mg/dl)	NT-proBNP (pg/ml)	S. Procalcitonin (ng/ml)	D-dimer (mcg/ml)	LDH (U/L)
3.57	206	3414.20	5.91	3.79	724

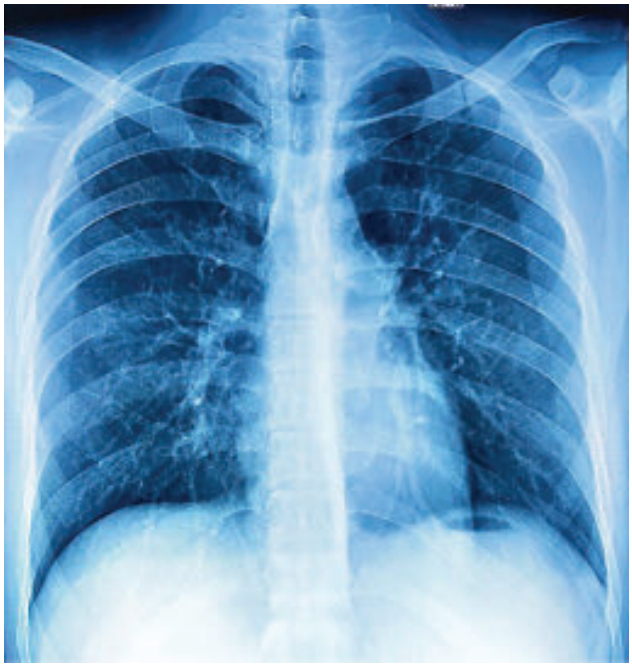

Fig.-2: *Clear lung fields on repeat chest x-ray P/A view, on discharge.*

After getting discharged, his 2weeks follow-up visit was uneventful and showed normal parameters of all blood works.

Discussion:

Typhoid fever is an acute systemic illness in which S. typhi get phagocytosed by RES in the small intestine, multiplies and then re-enters the bloodstream causing recurrence of bacteremia which coincides with the manifestations directly related to different organ involvement, local lesions to gut, endotoxemia or allergic reaction, and to treatment. But of the many respiratory complications, the ARD Shas been documented very rarely, out of which all have been reported to happen in young adults[3].

The characteristic pulmonary endothelial damage in ARDS usually requires the action of neutrophils, butit can also occur without pulmonary neutrophil infiltration, and in the setting of severe neutropenia-which is a feature of enteric fever[4]. Early diuretic therapy in ARDS in critically ill patients has better clinical outcomes, but is not conclusively known to have mortality benefit.

In 90% of patients, LFT is altered which returns to normal values after recovering, in virtually all. Endotoxemia is also present, which can be detected by the limulus test. This endotoxin can impair bile secretion, andthus may contribute to the pathogenesis of hepatic dysfunction during typhoid fever[5].

Hematological changesare common that include anemia, leucopenia, eosinopenia, thrombocytopenia, sub-clinical disseminated intravascular coagulation, and bleeding diathesis. The bone marrow study shows myeloid maturation arrest, decreased number of erythroblasts and megakaryocytes with increased phagocytic activity of histiocytes[6]. If GI bleeding is superimposed with reduced erythroblastosis, severe anemia can ensue, which we have observed in this case.

The neuropsychiatric manifestation of typhoid fever is not that common but has wide ranges- delirium, semi-coma, coma, meningism, meningitis, convulsions, generalized myoclonus, deafness, hemiplegia, infra-nuclear facial palsy, transient parkinsonism, and generalized transient hyper/ hypotonicity. Out of all these, delirium is the most common one[7].

In most of the cases the diagnosis is made empirically by clinical features, but culture and sensitivity of blood or bone marrow is the gold standard for diagnosing and providing treatment[8].

The antibiotic sensitivity pattern is changing and drug resistance is a burning issuethat is emerging with indiscriminate use of antibiotics[8].Changing patterns of both MDR (resistant to all three traditional first–line agents: Chloramphenicol; Ampicillin; and Co–trimoxazole) and Fluoroquinoloneresistant strains have been associated with reported changes in the severity and clinical profile of enteric fever[1].

Conclusion:

Our patient developed complications affecting major systems [respiratory, GI, hepatobiliary, hematologic, and neuropsychiatric within very short span of time; despite being on culture-sensitive antibiotic coverage with proper dose. The disease course here was erratic and the treatment protocol of MDR enteric fever favorably got him recovered. Having proper clinical knowledge and the rightful implication of this- is basically a life-saving tool in clinical practice; no matter what the lab report says.

Abbreviations:

BP: Blood pressure.RR: Respiratory rate.LFT: Liver function test.ICU: Intensive care unit.MDR: Multi drug resistant,NRBM: Non re-breather mask,MODS: Multi-organ dysfunction syndrome.ARDS: Acute respiratory distress syndrome;PCWP: Pulmonary capillary wedge pressure

Conflict of Interest:

The author stated that there is no conflict of interest in this study

Funding:
No specific funding was received for this study.

Ethical consideration:
The study was conducted after approval from the ethical review committee. The confidentiality and anonymity of the study participants were maintained.

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