Case Report

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Respiratory Distress in Pediatric Dengue Patients: Coinfections, Diagnosis Challenges and Treatment Strategies in an Endemic Setting

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ABSTRACT

Background: Respiratory distress complicating pediatric dengue fever cases underscores the significance of timely diagnosis and treatment, especially in endemic settings like Bangladesh. Coinfections with respiratory pathogens add to the complexity of management and treatment. This study aims to shed light on the coinfection and clinical implications of coinfections in pediatric dengue patients.

Case Presentation: We present a case report of two children with dengue fever who were diagnosed with coinfections, including Salmonella, Acinetobacter species, and Staphylococcus hominis. These patients exhibited severe clinical manifestations (Persistent fever, respiratory distress), and positive dengue serology results. Diagnostic workup revealed coinfections, highlighting the need to consider alternative pathogens in dengue patients.

Conclusion: Coinfections in dengue patients present unique challenges due to overlapping clinical features and the potential for delayed or missed diagnoses. The endemic nature of salmonella and dengue in Bangladesh increases the likelihood of co-occurrence. Understanding these associations is crucial for accurate diagnosis and timely intervention.

KEY WORDS
Bacteria; Bacteremia; Coinfection; Dengue.

INTRODUCTION

Infectious infections are a leading source of death and illness in underdeveloped countries like Bangladesh.[1,2] Many different combinations of co-infections have been documented in tropical regions.[3] Dengue fever, a virus spread by mosquitoes, has recently become a major cause of death and illness in Bangladesh The Ministry of Health and Family Welfare (MOHFW).[4] recorded 52,807 instances of dengue fever and 230 deaths from the disease between January 1 and November 20, 2022.[5]

As a result, clinicians face difficulty making a diagnosis of co-infections. Co-infections can be deadly if not treated quickly. Due to the endemic nature of both salmonella and dengue in Bangladesh, it is feasible for a child to contract both infections.[6] Again, Coagulase-negative Staphylococcus hominis infects the circulation and frequently forms biofilms on medical devices.[7] Co-infections with other infectious bacteria are also possible, albeit they are seldom recorded. Coinfections in dengue patients may occur due to the following factors:

i) the coincidence of two or more infections in countries where infectious diseases are ubiquitous

ii) the effect of the dengue virus on the immune system, which predisposes to other infections[8]

iii) pathology in some organ systems caused bydengue virus, which predisposes to superimposed infections.[9]

Here we describe a case series of dengue in two young children with coinfections with Salmonella enterica serovar enteritidis, multiple coinfections with Acinetobacter species, and Staphylococcus hominiswho developed a persistent fever, diarrhea, low platelet counts and a positive dengue serology test.

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CASE PRESENTATION-1

On September 6, 2022, a young boy, aged two years and four months, was brought to the emergency room at Ever Care Hospital in Dhaka, Bangladesh, complaining of a fever and a cough. Since September 9, 2022, he's had loose stools and vomited. On September 9, 2022, dengue NS1 was positive with thrombocytopenia of 63,000 cu mm, but dengue for IgG and IgM was negative. The patient was admitted to the hospital with a temperature of 98.7 degrees Fahrenheit, a blood pressure of 80/50 mmHg, a pulse rate of 120 beats per minute with low volume, a respiratory rate of 30 beats per minute, and a SpO2 of 100% in the room atmosphere. He also had some dehydration. When the chest was checked, there was only a vesicular breath sound. There were no signs of meningitis or brain injury. Later, he underwent a different investigation (Table I) and was diagnosed with dengue fever, Acute Respiratory Infection (ARI) and invasive diarrhea. Following admission, he received nebulization, Syp. fexofenadine, zinc oxide ointment, paracetamol, and I/V fluid bolus of normal saline and fluid for hydration, in addition to the injections of Ciprofloxacin, ondansetron and esomeprazole that were administered. A continuous 20% human albumin drip was started and lasted till the following night due to low blood pressure the following day (70/40 mmHg on September 10, 2022). Injected ceftriaxone was started on September 13, 2022, after stool C/S indicated the growth of Salmonella enteritidis (Table II). On the next day, his loose motion improved, his vital signs remained stable and he became afebrile. He was discharged from the hospital with medicine on September 14, 2022, with instructions to follow up with the Outpatient Department (OPD).

CASE PRESENTATION-2

A 6-year-old boy resident in Dhaka was admitted to Pediatric Intensive Care Unit (PICU) of Dr. Sirajul Islam Medical College and Hospital on 24 September 2022 with complaints of fever for 5 days with respiratory distress associated with abdominal pain, and a history of blackish stools. The patient was admitted to the hospital with a temperature of 101 degrees Fahrenheit, dyspnea, chest discomfort, an oxygen saturation of 85% in the room, a pulse of low volume and feeble, a heart rate of 170 beats per minute, a respiratory rate of 64 beats per minute and a blood pressure of 60/30 mmHg. Moreover, on admission, his abdomen was distended while examination. Then the diagnosis of dengue expanded syndrome was made. Ultrasonographic findings showed hepatomegaly, bilateral pleural effusions and mild ascites. Several investigations were done (Table I) such as CBC, CRP, serum electrolytes, SGPT, SGOT, D-Dimer, urine microscopy and antibiotic sensitivity test (Table II). The patient received four units of whole blood, three units of Fresh Frozen Plasma (FFP) and two units of apheresis platelets. Patient was kept Nothing by mouth (NPO) and received infusions of 5% DNS, 1% KT, 1% dopamine, 1% meropenem, 1% vancomycin, 1% ciprofloxacin and 2% human albumin. To treat pneumonia, 1% meropenem and 1% vancomycin were given. He was discharged from the hospital with the relatively stable condition on 2nd October, 2022, with medicine and instructions to follow upwith the OPD.

Table I Comparison of Investigation

Sl. No.	Test	Patient-1	Patient-2	Normal Value
01	COVID-19 Antigen Test	Negative	-	
02	Hemoglobin (Hb) (g/dL)	11.9→12.7→11.9→ 11.9→12.1→11.4	10.30→8.90→10.40→ 11.10→12→12→13.30	11-14
03	RBC count (10^12/L)	4.39→4.69→4.38→ 4.37→4.46→4.22	3.88→3.41→3.93→4.13→ 4.42→4.44→ 4.84	4.5-6.5
04	PCV (Packed Cell Volume) (%)	35.70→38.90→37.10→ 36.60→37.30→35.20	28.60→25.70→30→32.30→ 35.20→35.20→39	40-52
05	MCV (fl)	81.30→82.90→84.70→ 83.80→83.60→83.40	73.70→75.50→76.30→ 78.20→79.60→	
			79.50→ 80.60	75-87
06	MCH (pg)	27.10→27.10→27.20→ 27.20→27.10→27	26.50→26.10→26.50→ 26.90→27.10→	
			27→27.50	24-30

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Sl. No.	Test	Patient-1	Patient-2	Normal Value
07	MCHC (%)	33.30→32.60→32.10→ 32.50→32.40→32.40	36→34.60→34.70→ 34.40→34.10→ 34→34.10	31-37
08	RDW-SD (fl)	40.30→42.70→43.70→ 43.80→43.60→42.80	13.50→13.90→14.10→1 4.30→14.50→ 14.40→15.40	39-46
09	RDW-CV (%)	13.60→14.20→14→ 14.30→14.30→14	-	11.6-14
10	WBC Count (10^9/L)	2.75→5.62→6.51→ 6.30→6.60→5.69	3.2→6.4→5.8→ 6→5.8→7.6→4.4	5-15
11	Platelet Count (10^9/L)	130→125→60→110→ 150→150	300→1100→500→540→ 100→1100→740	150-400
12	Neutrophil (%)	28.30→36.80→40.90→ 36.20→31.90→37.40	43→42→29→ 24→29→32→24	20-50
13	Lymphocytes (%)	68→58.70→54.10→ 58.40→61.20→48.30	43→52→61→65→ 59→55→65	40-75
14	Monocytes (%)	3.30→4.10→0.20→4.90→ 5.00→8.30	06→04→06→ 8→9→10→09	2-8
15	Eosinophils (%)	0.40→0.20→0.20→ 0.50→1.70→6	3→2→4→3→ 3→3→2	1-6
16	Basophils (%)	0→0.20→0.20→0.00→ 0.20→0	0→0→0→0 →0→0→0	0-1
17	Absolute Neutrophil Count (10^9/L)	0.78→2.07→2.67→2.28→2.11→2.13	-	1.5-8
18	Absolute Lymphocytes Count (10^9/L)	1.87→3.30→3.52→3.68→4.04→2.75	-	6-9
19	Absolute Monocytes Count (10^9/L)	0.09→0.23→0.30→0.31→0.33→0.47	-	0.2-1
20	Absolute Eosinophils Count (10^9/L)	0.01→0.01→0.01→0.03→0.11→0.34	-	0.1-1
21	Absolute Basophils Count (10^9/L)	0→0.01→0.01→0→0.01→0	-	0.01-0.1
22	MPV (fl)	11.30→12→11.90→11.30→11.40→10.80	-	8.8-11.3
23	PCT (%)	0.08→0.08→0.02→0.09→0.09→0.10	-	0.19-0.39
24	PDW (fl)	13.40→18.30→9.30→14→16.80→14.90	-	9.3-14
25	C-Reactive Protein (CRP) (mg/dl)	<0.334	-	<0.33
	S. Electrolytes		Not Done	
26	S. Sodium (Na)- mmol/L	136	126→136→136→135→137→138→139	135-145
27	S. Potassium (k)- mmol/L	4.2	3.4→3.3→3.1→3→3.3→3.3→3.8	3.5-5
28	S. Chloride (Cl)- mmol/L	104	89→95→95→96→98→99→98	98-108
29	S. Bicarbonate (HC03) - mmol/L	22	-	24-32
30	S. Albumin (gm/dl)		2.3→4.2	3.4-5
31	S. Calcium (gm/dl)		7→7.8→7.9→8.8	8.1-10.4
32	Stool Routine Microscopy		Not Done	
	Color	Greenish	-	
	Consistency	Loose	-	
	Mucus	+	-	
	Blood	Nil	-	
	Pus Cells	Numerous	-	
	RBCs	0-2	-	
	Macrophages	Nil	-	

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Sl. No.	Test	Patient-1	Patient-2	Normal Value
	Parasites	Not found	-	
	Vegetable Cells	Nil	-	
	Yeast Cells	Nil	-	
	Fat Droplets	Nil	-	
	Epithelial Cells	Nil	-	
33	Rota Virus and Adenovirus in Stool		Not Done	
	Rota Virus	Negative	-	
	Adenovirus	Negative	-	
34	ABO and Rh typing	O Positive		
35	Aerobic C/S stool		Not Done	
	M	198	-	
36	Prothrombin Time (Seconds)	11.3	25→13	9.8-12.1
37	INR	0.94		-
38	Fibrinogen level (mg/dl)	230.7		180-350
39	APTT (Sec)	-	97→60→49→53→42	
40	S. Ferritin (ug/L)	-	>3000→>3000→939→517	24-336
41	Procalcitonin (ug/L)		25.93	0-0.05
42	SGPT (IU/L)	64	1008→485→327→272→242→204→103	7-56
43	SGOT(IU/L)	-	64→451→353→248→125	
44	D Dimer (mg/L)		>10→>10→5.15→2.11	<0.50
45	Urine RME (Physical Examination)	Not done		
	Color	-	Dark Brown	Colorless, pale yellow or straw
	Appearance	-	Strongly Turbid	Clear
	Specific Gravity	-	1.028	1.002-1.030
	Chemical Examination	-		
	Reaction	-	6	4.5-8
	Protein	-	+	Negative
	Glucose/ Sugar	-	Negative	Negative
	Ketone Bodies	-	+	Negative
	Bilirubin	-	+	Negative
	Urobilinogen		Normal	Normal/ Not increased
	Nitrate		Negative	Negative
	Leukocyte esterase		Negative	Negative
	RBC		0-1	0-2
	Pus Cells		1-3	0-5
	Epithelial Cells		0-1	0-10
46	Screening Test	Not Done		
	VDRL		Non-Reactive	
	HBsAg		Negative	
	HIV 1 & 2		Negative	
	HCV		Negative	
	MP		Negative	

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Table II Comparison of Antibiotic Sensitivity

SL No	Antibiotic	Patient-1 Salmonella ser. Enteritidis	Patient-2 Acinetobacter Species	Staphylococcus hominis
1	Ampicillin	R	-	-
2	Cefixime	R	-	-
3	Ciprofloxacin	R	R	S
4	Tetracycline	R	-	-
5	Trimethoprim / Sulfamethoxazole	R	-	-
6	Amikacin	-	R	-
7	Cefepime	-	R	-
8	Cefoperazone/ Salbactum	-	R	-
9	Ceftazidime	-	R	-
10	Ceftriaxone	-	R	R
11	Colistin	-	S	-
12	Gentamicin	-	R	S
13	Imipenem	-	I	-
14	Meropenem	-	S	-
15	Piperacillin+ Tazobactam	-	R	-
16	Tigecycline	-	S	S
17	Amoxiclav	-	-	S
18	Clindamycin	-	-	S
19	Doxycycline	-	-	S
20	Erythromycin	-	-	R
21	Levofloxacin	-	-	S
22	Linezolid	-	-	S
23	Oxacillin	-	-	R
24	Penicillin G	-	-	R
25	Rifampin	-	-	S
26	Teicoplanin	-	-	S
27	Vancomycin	-	-	S

DISCUSSION

The respiratory system can be significantly affected in dengue fever cases, especially when complicated by coinfections. In our case series, both patients exhibited respiratory distress requiring medical intervention. Respiratory symptoms in dengue fever can range from mild manifestations like cough and sore throat to severe complications such as pleural effusion and Respiratory Distress Syndrome (RDS). These respiratory complications can be exacerbated by concurrent bacterial or viral infections, as seen in our patients who presented with acute respiratory infections alongside dengue fever.One of the primary concerns with respiratory involvement in dengue cases is the potential for fluid accumulation in the lungs, leading to respiratory distress. This fluid accumulation can result from increased vascular permeability, a hallmark of severe dengue infection. Additionally, pleural effusion, as observed in Case 2, can further compromise respiratory function and necessitate aggressive management strategies.

Dengue fever epidemics occur annually in Bangladesh, a tropical nation, because of the country's high population density, unplanned urbanization, hot and humid climate, frequent rains during the monsoon season, environmental degradation and an inadequate supply of sanitation services,[10] Again, Acinetobacter is a member of the gram-negative, opportunistic coccobacilli bacterial family that is linked to a wide range of hospital-acquired infections. It is frequently transmitted to patients by remaining persistent on environmental surfaces and briefly colonizing healthcare personnel's hands.[11,12] Moreover, salmonellosis, an infection spread through contaminated food, is a serious global health problem. Mild to severe gastroenteritis, which can be fatal, can result from eating foods contaminated with Salmonella spp.[13]

Salmonella, including MDR strains, continues to be one of the primary bacterial foodborne causes of death, particularly in LMICs.[14] Recent years have seen a rise in the prevalence of multidrug-resistant Salmonella (MDR Salmonella), which includes resistance to clinically relevant antimicrobials such as fluoroquinolones and third-generation cephalosporins. Salmonella twhichis resistant to first-line antibiotics like ampicillin, chloramphenicol and trimethoprim-sulfamethoxazole is said to be resistant to multiple drugs. This has given rise to a new concern all over the world.[14] In our case 2, he was resistant to various drugs, including ampicillin, cephalosporin, Ciprofloxacin, tetracycline, and trimethoprim-sulfamethoxazole.

Staphylococcus hominis is a member of the Staphylococci genus that is gram-positive and coagulase-negative. Just like the vast majority of other coagulase-negative Staphylococci, S. hominis is known to cause a wide variety of nosocomial, or hospital-acquired, infections. It also has the potential to cause infection in patients whose immune systems are abnormally compromised.[15]

Infection with the dengue virus has been shown to co-occur with the presence of certain microorganisms. Among them were, Escherichia coli, Salmonella sp., Streptococcus pneumoniae, Mycobacterium tuberculosis, Mycoplasma pneumoniae, Shigella sonnei, Klebsiella pneumonia, Klebisellaozaenae, Enterococcus faecalis, Moraxella lacunata, Staphylococcus aureus, Rosemonas sp., Haemophilus influenza, Candida tropicalis and herpes viruses.[16] One

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possible reason for these co-infections is damage to the digestive epithelial barrier, which could be caused by endothelial damage or intestinal bleeding. This would allow pathogens from the digestive system to get into the bloodstream. In fact, intestinal flora microorganisms seem to be the most common in these cases. Also, physiopathological changes in the vascular and blood-clotting system that can be seen in some organs or systems may make infections worse. Lastly, a bacterial infection happening at the same time as a dengue virus infection could be a simple coincidence of time or, more likely, could be caused by the virus, which is thought to weaken the immune system.[17] Although co-infections of dengue and Salmonella Enteritidis and multiple coinfections with Acinetobacter species and Staphylococcus hominis have been reported in a small number of adult patients, there have been even fewer cases in children. Coinfections with bacterial pathogens, such as Salmonella enteritidis, Acinetobacter species, and Staphylococcus hominis, add another layer of complexity to respiratory management in dengue cases. These coinfections can exacerbate inflammation and compromise lung function, leading to more severe respiratory symptoms and increased mortality risk.

CONCLUSION

Rapid diagnosing respiratory distress in pediatric dengue cases with coinfections is crucial for prompt intervention. Clinicians should prioritize respiratory assessment and tailored antimicrobial therapy to improve outcomes in endemic regions like Bangladesh.

Consent

The patient's parents had written informed consent taken for publishing this case report as well as images, because of patient not adult.

DISCLOSURE

All the authors declared no competing interest.

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