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CASE REPORT

ATYPICAL PRESENTATION OF TWO CHIKUNGUNYA CASES: A CASE SERIES DURING 2025 OUTBREAK

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Abstract

Bangladesh is experiencing a sharp rise in viral infections, particularly during the current monsoon season. Dengue, chikungunya, and COVID-19 are now posing a triple threat to public health. As their symptoms are overlapping, a strong diagnostic support is pivotal in severe cases.Here, we describe two older male chikungunya virus patients who, in addition to their typical symptoms, have significant organ involvement. Pneumonia caused by chikungunya affecting the lungs occurred in the first patient. The second case had similar lung involvement with encephalitis. Both had diabetes and hypertension, two co-morbidities that increase the chance of developing serious illness. The second case was also a known case of chronic kidney disease (CKD), old cerebrovascular disease (CVD) and ischaemic heart disease (IHD). He was transferred to the critical care unit and eventually passed away. These cases demonstrate how crucial it is that Chikungunya PCR tests be made available across the country at a reasonable cost in order to prevent needless treatment and investigations as well as early case diagnosis.

Keywords: *Chikungunya, Pneumonia, Encephalitis*

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Introduction:

Chikungunya virus (CHIKV) infection is a neglected tropical disease, caused by an RNA virus (genus: Alphavirus; family: Togaviridae. With the spread of Aedes mosquito vectors, chikungunya is therefore regarded as a significant re-emerging public health issue in both tropical and temperate nations. The disease was first described in Tanzania in 1952, and the virus was first isolated in Thailand in 1958. The term “chikungunya” refers to the twisted posture of infected individuals who experience excruciating joint pain and is derived from a word in the southern Tanzanian Kimakonde language that means “that which bends up”[1]. This RNA virus is known to have three genotypes: West African, East/Central/South African (ECSA), and Asian[2]. In the Americas, there were more than 2.6 million probable cases of chikungunya by the end of 2017. Since then, the virus has spread around the world, causing occasional outbreaks and sporadic cases in Asia, the Americas, and Africa. In two recent outbreaks more than 50,000 suspected cases were found in Ethiopia in 2019 and 160,000 in Paraguay in 2023. In 2024 and 2025multiple outbreakswere reported on the island of Réunion in the Indian Ocean[3]. Bangladesh is habituated in facing periodic outbreak of dengue fever, but chikungunya outbreak is rarely seen. It may be because of the prolonged presence of antibody and cross immunity against genotypes[4]. The last known outbreak of chikungunya happened between April 1, 2017 to Sept 7, 2017, and the Bangladeshi Ministry of Health reported 984 cases confirmed by real-time PCR assay. More than 13176 clinically confirmed cases in 17 of 64 districts[5]. Apart from typical presentation in acute phase and chronic debilitating arthritis may also be seen.Some systematic manifestations like cardiac complications was reported in 37% (myocarditis and arrhythmia)[6,7], neurological complications has been reported in 16-25% of patients. These include encephalitis, facial paralysis, sensorineural deafness, and Guillain-Barré syndrome (GBS)[7–9]. Upto 17% reports of pneumonia with 8% respiratory failure was also reported[7], and rarely, ocular complications (conjunctivitis, optic neuritis, iridocyclitis, episcleritis, retinitis and uveitis)[10] has also been reported. Herein, we will discuss two elderly male patients with multiple co-morbidities who had with chikungunya fever with atypical presentation that wasseldom reported before.

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Both Chikungunya cases were confirmed by VIASURE Mosquito-Borne (Zika, Dengue and Chikungunya virus) multiplex RT-PCR detection Kit (Spain). This test is performed using the Rotor-Gene Q PCR system by Qiagen, Germany. The clinical specificity of this PCR kit is 100% and the analytical sensitivity is up to 20 Copies/reaction.

Case 1:

Chikungunya and bilateral pneumonia, a rare presentation

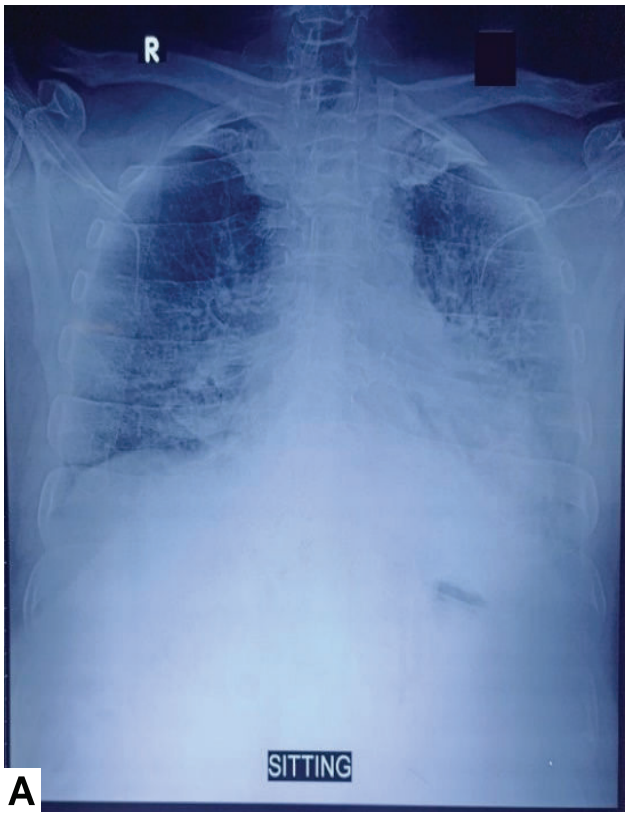
A 64-year-old diabetic, hypertensive male, got admitted to our hospital with the complaints of fever for 3 days, which was high grade, highest recorded temperature was 105°F which was not associated with chills and rigor. Fever was preceded by frontal headache for one day. He also developed severe ankle and low back pain which made him unable to walk without limping or support. On query he further complained of intermittent dry cough for 2 days. There was also history of dengue fever twice before. He was not vaccinated against influenza or pneumococcus before. He took oral hypoglycemic medications (Gliclazide 30mg, metformin 500mg, empagliflozin 5mg) for diabetes control. Beta blocker (bisoprolol 2.5 mg), calcium channel blocker (amlodipine 5mg) with angiotensin receptor blocker (olmesartan 20mg) all in once daily dose for hypertension. He was compliant to medications and maintained regular follow up with good control of comorbidities. There was no history of asthma, osteoarthritis or other connective tissue disease. On the day of admission, he had flushed face, temperature was102°F, pulse84 b/min with regular rhythm & volume, blood pressure :110/70mmHg, SpO2: 98% in room air, There was grade II tenderness and swelling of both his ankle joints. There was no anemia, jaundice, oedema, dehydration or clubbing. Precordium, abdomen and lungs were unremarkable on examination. No conjunctival congestion or rash. He was kept on conservative management and his anti-diabetic and antihypertensive medications were kept on hold. Dengue-chikungunya-zika multiplex PCR was sent (detected Chikungunya) along with other lab parameters (Table 1). On his peripheral blood film there

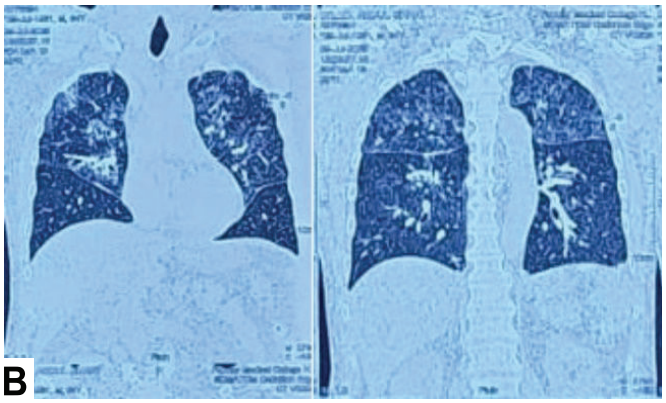
Table I

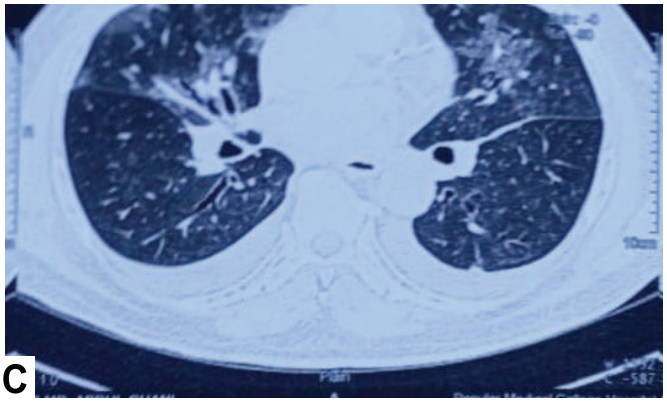
Clinical scores and laboratory parameters of the case 1

Clinical and Lab Parameters	Normal range	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
qSOFA	0	1	0	0	0	0	
NEWS	2	11	6	3	0	0	
MEWS	3	4	1	1	0	0	
Hemoglobin(g/dL)	13-17	10.6	10	11.2	12.9	12.4	12.4
HCT %	39-51	31.5	31	33.6	39.3	38.2	37.5
Total WBC(k/µL)	4-10	4.31	3.4	5.5	5.6	6.4	6.74
Platelet Count(k/µL)	150-410	70	90	50	50	40	50
N:L ratio		8	8.2	8.6	4.9	6.9	12.6
ESR (mmHg 1St hour)	0-14	09	07	10	14	05	10
SGOT (U/L)	<40	23				57	
SGPT (U/L)	<41	17				32	
CRP (mg/L)	<5	8.85		50.12		10.37	
S. Creatinine (mg/dL)	0.7-1.2		1.13				
S. Sodium(mmol/L)	136-145		123			133	134
S. Potassium(mmol/L)	3.5-5.1		4.3			3.9	4.35
S Chloride(mmol/L)	98-107		95			100	102
S. Bicarbonate(mmol/L)	24-30		20			20	
D-Dimer(µg/mL)	<0.5		1.46			1.28	
Ferritin (ng/mL)	21-274					1730	
Procalcitonin(ng/mL)	<0.5 sepsis unlikely		0.16			0.05	
Cardiac Troponin I (ng/mL)	<0.034		0.016				
NT pro BNP (pg/mL)			2548				
pH			7.42	7.37			
pCO2 (mmHg)			28.5	22.7			
pO2 (mmHg)			35.3	80.8			
HCO3 (mmol/L)			20.1	16.1			
PO2/FIO2			168	384.7			

Day 1 signifies first day of admission; qSOFA: quick sequential organ failure assessment; NEWS: National early warning score; MEWS: Modified early warning system; HCT: hematocrit; WBC: White blood cells; N:L ratio (neutrophil lymphocyte ratio), ESR: erythrocyte sedimentation rate; SGOT: serum glutamic oxaloacetic transaminase test; SGPT: Serum glutamic pyruvic transaminase; CRP: C reactive protein; NT pro BNP: N terminal pro B type Natriuretic Peptide.







Investigation for:	Respiratory Panel.	Test Method:	Real Time PCR [Rotor-Gene Q, QIAGEN, GERMANY]
Pathogen Type	**Pathogen Name**		**Genetic Material**
Virus	*Influenza A*		Not Detected
	Influenza A-H1		Not Detected
	Influenza A-H1pdm09		Not Detected
	Influenza A-H3		Not Detected
	Influenza B		Not Detected
	Respiratory Syncytial Virus A & B		Not Detected
	Rhinovirus/Enterovirus		Not Detected
	Metapneumovirus		Not Detected
	Parainfluenza Virus 1		Not Detected
	Parainfluenza Virus 2		Not Detected
	Parainfluenza Virus 3		Not Detected
	Parainfluenza Virus 4		Not Detected
	Bocavirus 1/2/3/4		Not Detected
	Coronavirus 229E		Not Detected
	Coronavirus NL63		Not Detected
	Coronavirus OC43		Not Detected
	Coronavirus HKU1		Not Detected
	Adenovirus		Not Detected
	SARS-CoV-2		Not Detected
Bacteria	*Bordetella pertussis*		Not Detected
	Chlamydophila pneumoniae		Not Detected
	Mycoplasma Pneumoniae		Not Detected
	Legionella pneumophila		Not Detected
	Streptococcus pneumoniae		Not Detected
	Streptococcus pyogenes		Not Detected
	Haemophilus influenzae		Not Detected
	Moraxella catarrhalis		Not Detected
	Internal Control (IC)		Detected

Comment: *No Pathogen is* detected.

The **NoeDx Respiratory-27 pathogen Detection Kit** containing all the reagents necessary for nucleic acid extraction, amplification, and detection of 27 bacteria and viruses (or their subtypes) that cause respiratory symptoms, including SARS-CoV-2. This test is performed using the **Rotor-Gene Q, QIAGEN, GERMANY.**

Specificity: The clinical specificity of this PCR kit is **100%.**

Sensitivity: The analytical sensitivity is **2.40** Copies/reaction (Varies by pathogens)

Note: False negative results can occur due to sample collection from nasopharyngeal swabs, improper or poor handling of specimens after collection or before testing, failed extraction, the presence of PCR inhibitors or technical errors. All these factors should be considered when interpreting test results.

D

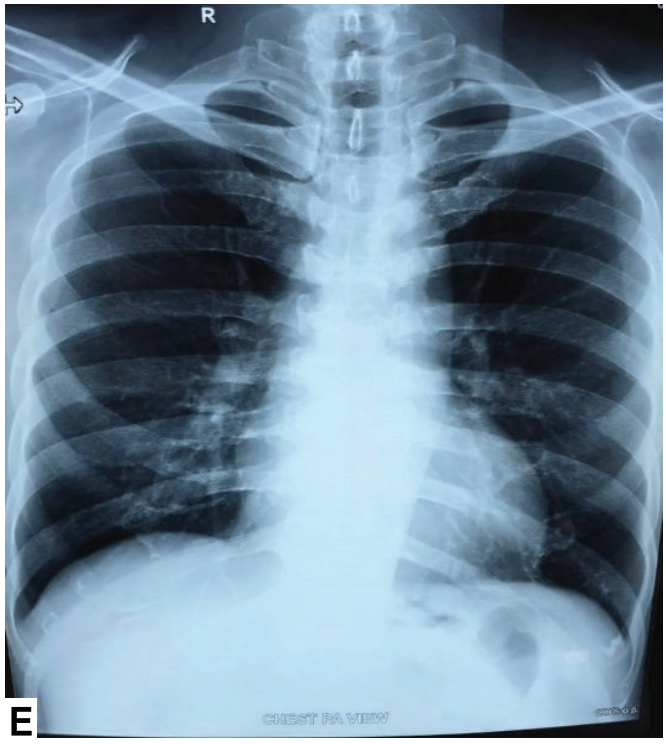


Figure 1: Chest X-ray of 1st case, (A) from day 2 of admission showing extensive bilateral infiltrates, (B) coronal and (C) axial cutof HRCT chest from day 3 : ground glass opacities in both upper lobe and right middle lobe, suggestive of alveolar infiltrates. Bilateral mild pleural effusion. (D) Respiratory panel- real time PCR: No pathogen. (E) Chest X-ray 4 days after discharge showing improved radiograph

was microcytic hypochromic anemia.

On second day morning, he developed restlessness, severe shortness of breath with worsening cough and mild mucoid expectoration but no orthopnea. There were bilateral coarse crepitations from mid chest. His SpO2 dropped to 75% on room air, respiratory rate was 44 breaths per minute, GCS 14. Blood pressure was 110/70 mmhg, Heart rate came down to as low as 46 beats per minute. He still had a temperature of 101°F. His saturation was maintained to 94% on 15L O2 (non-rebreathable mask). Immediate Chest Xray was done at bedside, which revealed bilateral lung infiltrates from mid to lower zone(FIG- 1A).Arterial blood gas showed marked hypoxia suggesting ARDS (Table I). ECG revealed sinus bradycardia, troponin-I, NT pro BNP and procalcitonin unremarkable. D-dimer was slightly raised. His HRCT chest non-contrast (Fig 1B,C) showed ground glass opacities in both upper lobe and right middle lobe, suggestive of alveolar infiltrates. Bilateral mild pleural effusion. Coronary artery calcifications were also noted. Bedside ECHO revealed No abnormality (Ejection fraction 62%). Before adding furthertreatment, we did a respiratory panel (Fig -1D, sputum for Grams's stain and culture sensitivity, Gene xpert and fungal culture which excluded all other pathogens. Blood culture came out negative.

He was managed with high dose intravenous hydrocortisone 200mg stat and 100mg 8hrly for Day 1, then gradually tapered off by day 5. He also received antibiotics (IV meropenem and oral moxifloxacin for 7 days), nebulization with budesonide with bronchodilators and symptomatic propantheline bromide for bradycardia. After initiating steroids his oxygen dependency came down to 8L on day 3. On day 4, he maintained on 2L with nasal cannula and became afebrile. Thereafter on day 5, he was free from oxygen. He was discharged on day 6 without any oxygen support. Four days after he came for follow up with normal CBC, negative dengue IgM/IgG antibodies and Chest radiograph was also normal (Fig- 1E).

Day 1 signifies first day of admission; qSOFA: quick sequential organ failure assessment; NEWS: National early warning score; MEWS: Modified early warning system; HCT: hematocrit; WBC: White blood cells; N:L ratio (neutrophil lymphocyte ratio), ESR: erythrocyte sedimentation rate; SGOT: serum glutamic oxaloacetic transaminase test; SGPT: Serum glutamic pyruvic transaminase; CRP: C reactive protein; NT pro BNP: N terminal pro B type Natriuretic Peptide.

Case 2:

Chikungunya encephalitis and respiratory failure

A 75-year- old diabetic, hypertensive male, known case of CKD, old CVD (11 years back) and IHD, complained of continued high-grade fever for 2 days. His highest recorded temperature was 105°F. Fever was associated with severe bodyache,increased pain in both knees, ankle and small joints of hand and feet. There was no cough, abdominal pain or vomiting. He developed altered consciousness the next day and admitted to this hospital for better management. After admission, we found him to be disoriented. Not obeying commands. GCS was 9 (E1M5V3). There was no neck rigidity. Kernig's Sign was negative. Pupil was bilaterally equally reacting to light. Temperature was 101°F, blood pressure was 130/70mmHg, heart rate was 100beats per minute, respiratory rate was 20 breaths per minute. He had mild bipedal edema. His Spo2 was 92% on Room air (99% on 1L o2). There was grade II tenderness over his both knee and ankle joints, but no swelling or significant erythema seen. Heart, lungs and abdomen was normal on examination. On investigation: His CBC was normal except mild anemia and thrombocytopenia (110k) and hyponatremia, serum creatinine was slightly high, arterial blood gas showed respiratory alkalosis (Table-II). Dengue NS1 antigen came out negative, Urine RME revealed trace albuminuria and sugar otherwise normal, Liver function test, Bedside Chest Xray and Echocardiography (EF: 61%) was normal. Multiplex PCR (Dengue-Zika-Chikungunya) detected chikungunya. CSF study was done which showed Clear fluid, total WBC was 03/Cumm (100% lymphocytes), high protein (74.9 mg/dL) and glucose (111.96 mg/dL), ADA was also normal (1.1 U/L). Gram's stain, AFB stain, Gene xpert for mycobacterium tuberculosis and culture was negative. CSF PCR for Neuro-9 panel was negative (Fig- 2A). CT scan of brain was done which revealed generalized cerebral atrophy and tiny hypodense lesion in left frontal lobe and left thalamus (Fig-2 C,D). MRI brain (with MRA) was also done which showed chronic bilateral periventricular ischaemic changes with mild generalized cerebral atrophy. He was on conservative management with NG tube feeding, intravenous paracetamol one gramfor fever and pain, oral correction of hyponatremia, high dose hydrocortisone injection and management of co-morbidities.

The following day of admission he developed shortness of breath which was progressive upto day 4 of admission. We had totransfer him tocritical care unit for better management. GCS was not improving, respiratory rate went up to 44 breaths per minute, and heart rate was 154 beats per minute. On auscultation of lungs there was bilateral coarse crepitation on both lungs, more on the right side. He was needing 15L on non-rebreathable mask to maintain 94% oxygen. On investigation (Table-2) total count WBC was normal throughout, thrombocytopenia was progressive upto day 3 then began improving. D dimer, procalcitonin and fibrinogen was not significantly high. Sputum culture, Gram's stain, AFB

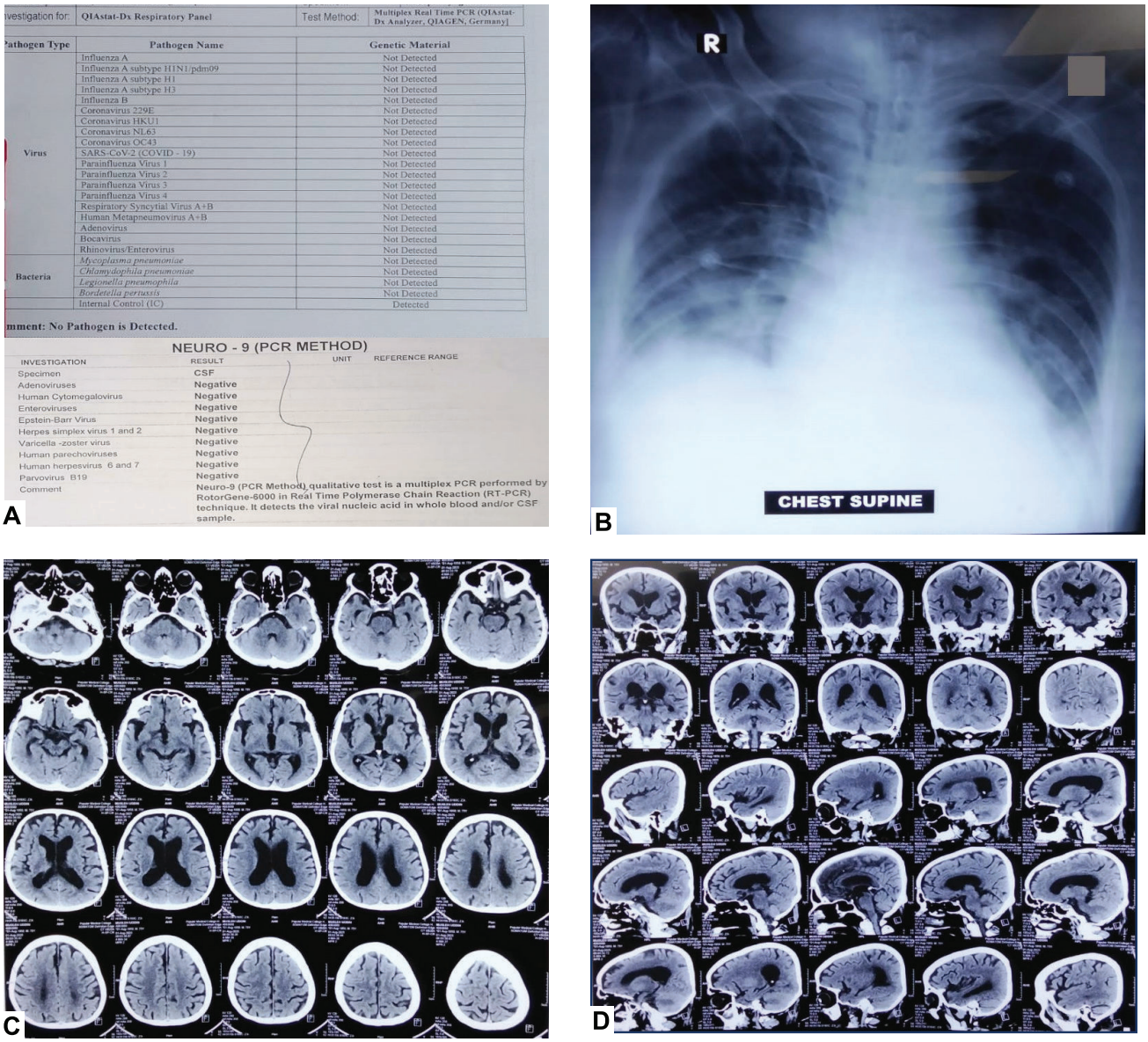
stain, fungal stain, Gene xpert for mycobacterium tuberculosis was negative. Blood culturealso showed no growth. NT pro BNP was high but bedside echo was normal. ECG showed only sinus arrhythmia. ABG revealed persistent hypoxia from day 4. On day 5, he developed metabolic acidosis due to deteriorated renal function. Respiratory panel from nasopharyngeal swab was unrevealing (Fig 2 A), There was bilateral lung infiltrates more on the right side (Fig-2B). He was Intubated on day 4 upon shifted to ICU, where he was managed with inj. meropenem 1g(12hourly), inj. dexamethasone 5 mg (8 hourly) with other conservative management. Unfortunately, his family members took him home after 4 days of treatment in ICU. He succumbed to death after 2 days at home.

Table II

Laboratory parameters of case 2

Lab Parameters	Normal range	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Hemoglobin(g/dL)	13-17	11.5	12.3	11.3	12.1	12.2	10.8	11.0
MCV (fL)	81-94	68.4	67.9	68.8	70.5	69.2	70.3	73.3
HCT %	39-51	36.4	39.1	37.3	40.3	40.3	36	37.9
Total WBC(k/µL)	4-10	6.95	6.5	3.67	4.99	4.99	7.02	7.89
Neutrophil (%)	40-80	87	80	72	80	80	81	82
Platelet Count(k/µL)	150-410	110	80	70	75	80	70	105
N:L ratio		12.4	5.3	3.1	5.3	5.3	5.4	6.3
ESR (mmHg 1St hr)	0-14					32	41	52
SGOT (U/L)	<40	23		28		30	77	53
SGPT (U/L)	<41	15		22		37		50
S. Albumin (g/dL)	3.5-5.2	3.62					3.09	
CRP (mg/L)	<5					55		193.4
S. Urea(mg/dL)	16.6-48.5	42.8		38	30.6	28	47.4	68.48
Uric acid (mg/dL)	3.5-7.2	7.30						
S. Creatinine (mg/dL)	0.7-1.2	1.77	1.62	1.55	1.47	1.672.13		1.91
S. Sodium (mmol/L)	136-145	132	133	139	138	136	140	
S. Potassium(mmol/L)	3.5-5.1	3.7	3.9	3.93	3.9	3.6	3.86	
S Calcium (mg/dL)	8.8-10.6	8.8					7.75	
S.Magnesium(mg/dL)	1.6-2.3	1.6					1.6	
D-Dimer(µg/mL)	<0.5				1.32			
Procalcitonin(ng/mL)	<0.5				0.45			3.22
Fibrinogen (mg/dL)	200-400				512			
PT (sec)/INR	12-17				17/1.47			
APTT(sec)					32			
S. Ammonia (µmol/L)	9-33			09			10	
Cardiac Troponin I (ng/mL)	<0.034	0.09	0.10		0.08			0.11
NT pro BNP (pg/mL)	<125				4054			
pH	7.35-7.45	7.5	7.4	7.5	7.45	7.36*	7.30*	7.34*	7.36*
pCO2(mmHg)	35-45	23.2	24.2	23.8	25	41.1	31.7	36.8	38.5
pO2(mmHg)	75-100	71.8	75.4	71.6	56.4	79.6	92.3	76.5	86
HCO3(mEq/L)	22-26	23.2	22.3	22.9	20.7	22.6	16.7	19.9	21.4

MCV : mean corpuscular volume, N:L ratio (neutrophil lymphocyte ratio), HCT: hematocrit; WBC: White blood cells; ESR: erythrocyte sedimentation rate; SGOT: serum glutamic oxaloacetic transaminase test; SGPT: Serum glutamic pyruvic transaminase; CRP: C reactive protein; NT pro BNP: N terminal pro B type Natriuretic Peptide. *after intubation.



Investigation for:	QIAstat-Dx Respiratory Panel	Test Method:	Multiplex Real Time PCR (QIAstat-Dx Analyzer, QIAGEN, Germany)
Pathogen Type	Pathogen Name	Genetic Material	
Virus	Influenza A	Not Detected	
	Influenza A subtype H1N1/pdm09	Not Detected	
	Influenza A subtype H1	Not Detected	
	Influenza A subtype H3	Not Detected	
	Influenza B	Not Detected	
	Coronavirus 229E	Not Detected	
	Coronavirus HKU1	Not Detected	
	Coronavirus NL63	Not Detected	
	Coronavirus OC43	Not Detected	
	SARS-CoV-2 (COVID - 19)	Not Detected	
	Parainfluenza Virus 1	Not Detected	
	Parainfluenza Virus 2	Not Detected	
	Parainfluenza Virus 3	Not Detected	
	Parainfluenza Virus 4	Not Detected	
	Respiratory Syncytial Virus A+B	Not Detected	
	Human Metapneumovirus A+B	Not Detected	
	Adenovirus	Not Detected	
	Bocavirus	Not Detected	
	Rhinovirus/Enterovirus	Not Detected	
Bacteria	*Mycoplasma pneumoniae*	Not Detected	
	Chlamydophila pneumoniae	Not Detected	
	Legionella pneumophila	Not Detected	
	Bordetella pertussis	Not Detected	
	Internal Control (IC)	Detected	

mment: No Pathogen is Detected.

NEURO - 9 (PCR METHOD)

INVESTIGATION	RESULT	UNIT	REFERENCE RANGE
Specimen	CSF		
Adenoviruses	Negative		
Human Cytomegalovirus	Negative		
Enteroviruses	Negative		
Epstein-Barr Virus	Negative		
Herpes simplex virus 1 and 2	Negative		
Varicella -zoster virus	Negative		
Human parechoviruses	Negative		
Human herpesvirus 6 and 7	Negative		
Parvovirus B19	Negative		
Comment	Neuro-9 (PCR Method) qualitative test is a multiplex PCR performed by RotorGene-6000 in Real Time Polymerase Chain Reaction (RT-PCR) technique. It detects the viral nucleic acid in whole blood and/or CSF sample.		

Figure 2 : A) Showing negative nasal swab for Respiratory panel (above) and CSF for Neuro-9 PCR panel, B) CXR supine view after respiratory distress showing bilateral (right >left) pulmonary infiltrates, C) &D) showing CT brain Axial and sagittal view respectively showing, tiny hypodense lesion in left frontal lobe and left thalamus generalized cerebral atrophy and ventriculomegaly

Discussion:

Reappearanceof chikungunya cases have been reported in Bangladesh in 2024, following a seven-year hiatus. From 19th October to 31st December 2024, 138 Chikungunya positive cases were reported. These cases where mostly male and over 30-years old.[11]. This is by far the fifth outbreak happening in Bangladesh. The first three were in 2008 (Rajshahi) , 2009 (Pabna) , 2012 (Tangail) and 2017 (Dhaka) respectively[12]. In 2025 the cases still continue to rise. Up to May 28th,2025 IEDCR alone reported 153 PCR confirmed cases out of 337 suspected patients from both Dhaka North and South City Corporation[13]. These numbers are just the tip of iceberg as there is lack of national surveillance data.

Pneumonia in Chikungunya is not a rare entity. About 17% patients presented with pneumonia in a report from 2005-2006 from Reunion[7]. In previous outbreak of 2017, from Dhaka Bangladesh, there was no pulmonary involvement reported in any of the 690 participants[14]. Khare et al. reported a similar case like ours in 2022, from India. He described an elderly male

with diabetes and hypertension suffering from bilateral consolidation. However,he had no pleural effusion like our case [15]. They had successfully treated the patient with steroids. There has been reports of co-infection of chikungunya with respiratory syncytial virus, influenza and adenovirus especially in elderly and diabetics [16] which was excluded in both of our cases by respiratory panel. Bacterial Infection was also excluded by sputum and blood culture. The pattern of lung involvement was different in both cases, upper lobe and perihilar region being more involved in case 1 and lower lobes, predominantly right lung being more involved in case 2. In both cases bilateral involvement was a common feature. How CHIKV infects the lung is still understudied. Presence of previous respiratory disease, co-infection with other respiratory viruses may predispose the condition. Additionally, viral respiratory infections mainly show up as a Th1 response, in which IFN-ã, CD8 T cells, and NKCs all contribute to viral clearance, a process that is also observed for CHIKV in other organs[17].

Our 1st case also suffered from bradycardia, which was resolved on follow up after discharge, may be a presentation of chikungunya myocarditis. We excluded that by ECG and cardiac enzymes [18]. He also showed delayed platelet recovery, even after 3rd afebrile day which may be explained by increased platelet activation due to the inflammatory process[19].

Encephalitis was reported to be the most common neurological presentation. In 2017, the largest cohort reported from Bangladesh documented 11 (out of 690 suspected cases) with neurological impairment. Most of these cases were in elderly age group, All of them except one presented with altered consciousness and diagnosed as encephalitis. Although PCR from CSF was not done in all cases. The common co-morbidities reported was diabetes, hypertension and less commonly CKD and IHD[20]. Other neurological manifestations were encephalopathy, GBS, myelitis, ADEM(Acute demyelinating encephalomyelitis), brainstem encephalitis, cranial neuropathy[21]. Unfortunately, wecould not do chikungunya PCR from CSF due to unavailability of test kit. But we excluded other possible viral (Neuro -9 PCR from CSF, Fig-2A) and pyogenic causes (by cytology, biochemistry and culture). Tuberculosis was also excluded by ADA and Gene xpert MTB/RIF. A similar case report in India has documented bilateral numerous small punctate lesions in the white matter with periventricular diffusion-restricted spots in MRI brain. That patient underwent intravenous immunoglobulin (IVIG) therapy for approximately 7 days[22]. Although there are reports of good response to IVIG therapy[23], more evidence base is needed to prove it's cost-effectiveness in a low income country like Bangladesh. In our encephalitis case, we only found cerebral atrophy and features of his old CVD in brain imaging. CHIKV appears to enter the brain by first infecting the brain vasculature's endothelial cells, which makes it easier for the brain parenchyma to become infected and may result in encephalitis. Cytokine profiling of neurologically ill patients during CHIKV infection revealed elevated levels of TNF-α, IFN-α, and IL-6 in CSF samples, which is indicative of a type I IFN response[17,24].

From the first outbreak of chikungunya in 2008 to the fifth one in 2025, chikungunya virus has changed its molecular characteristics. The evolution and emergence of a novel sub-lineage within the previously prevalent ECSA genotype of the virus in Bangladesh was demonstrated by recent phylogenetic research in late 2024.Given the strong kinship between these outbreak strains, it is probable that their most recent common ancestor developed from the local strains that were previously in use in early 2019[11]. Unfortunately, we are still relying on 2017 national guidelines which lacks the treatment outline of organ involving severe cases. We need urgent measures to learn from our experience in this outbreak, generate evidence base for rational treatment of atypical presentations of chikungunya fever and update management plan to avoid fatalities and implement prompt management throughout the country.

Conclusion:

The actual burden of the chikungunya fever is underestimated because there is no regular national surveillance system for Chikungunya, diagnostic capacity is low, and there is clinical overlap with Dengue, especially during the monsoon season. Even if the acute infection resolves on its own, if there is neurological and respiratory involvement, it could become fatal. Preventive actions are encouraged for the general population to minimize mosquito breeding grounds and prevent mosquito bites, especially in and around dwellings.

Conflict of Interest:

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

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Consent for publication:

Informed written consent was taken from the parents of the patient to publish details relevant to the disease and management.

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