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CASE SERIES OF LASSA FEVER PATIENTS WITH ADVERSE OUTCOMES DESPITE HIGHER CT VALUES

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This study was a collaborative effort of the authors. The authors reviewed and approved the final version of the manuscript for publication.

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ABSTRACT
Lassa fever is endemic to West Africa. It is associated with an acute hemorrhagic illness. It is diagnosed through reverse transcription polymerase chain reaction (RT-PCR), in which low cycle threshold (Ct) values correspond to high viral load. This paper describes a case series of two patients in Owo, Ondo State, Nigeria (Infection Control and Research Centre, Federal Medical Centre), a National Reference Centre for viral hemorrhagic fever. These cases were confirmed by RC-67 and RC-69 Lazzaro, RC-67, and RC-69 Lazzaro, and RC-67 and RC-69 Lazzaro. We collated clinical and laboratory data as well as the sociodemographic data of the two patients. Both patients were treated in accordance with the guidelines, which include the use of intravenous ribavirin, supportive therapy, and transmission. The first case was a 45-year-old male with CT values of 32.817 (G) and 31.374 (L) who suffered acute kidney injury, haemorrhage, and died 8 days after admission. In the second scenario, a 37-year-old male patient presented with acute kidney injury and viral encephalitis, and was unable to survive 4 days post a session of hemodialysis. His Ct values of 35.88 and 36.25 could suggest the possibility of a poor clinical outcome, albeit the higher Ct value. This shows that viral load is not a complete determinant of the illness's severity. Other factors, such as host attributes, viral heterogeneity, immune dysregulation, possibly late treatment, and others, could significantly influence the illness's course and outcome.

Keywords: Lassa fever patients' Cycle threshold, Adverse Outcomes

1.0 INTRODUCTION

Lassa fever is an acute viral hemorrhagic fever caused by the *Lassa mammarenavirus*. LASV is an RNA virus belonging to the *Arenaviridae* family (Olasoju *et al.* 2024; Malik *et al.* 2023; Besson *et al.* 2024; Aripov *et al.* 2022). Most cases occur as a result of direct contact with food and other household items contaminated with the urine and

faeces of *Mastomys natalensis.* This species of rat is the multimammate rat, and it is the primary host. In Nigeria, which is one of the endemic countries, such rats are very abundant (Akafa *et al.* ., 2025; Olasoju *et al.* ., 2024; Besson *et al*., 2024; Aripov *et al*., 2022). Lassa fever can also be spread via direct infected person contact, especially within the healthcare setting, which is responsible for 20% of all Lassa fever transmissions (Besson *et al*., 2024). Nigeria also had disruptions in Lassa fever public health records in 2018 due to an outbreak (Siddle *et al.*, 2018). Lassa fever can be asymptomatic, mildly febrile, or present with severe illness, high morbidity and mortality (Saka *et al.* ., 2025; Malik *et al.* ., 2023; Owhin *et al.*, 2023). Lassa fever can also have. In mild cases, this can range up to 15% and in severe cases 60%, and can even reach 90-100% when treatment is not provided in time (Asogun *et al.*, 2016; Saka *et al.*, 2025; Malik *et al* ., 2023). The first signs and symptoms of Lassa fever are vague and nonspecific. This may lead to mistreatment of the patient for malaria and typhoid fever. This may lead to a missed opportunity for earlier treatment of Lassa fever and an increased risk of mortality due to delayed treatment.

Administration of the antiviral ribavirin early in the course of treatment improves survival; thus, delays in beginning treatment harm survival (Asogun *et al.*, 2016; Okogbenin *et al.*, 2022). The pathophysiology of Lassa fever remains largely unknown; however, hyperinflammation may be one of a number of unaddressed pathophysiologic phenomena that contribute to the overall clinical picture (Okogbenin *et al.*, 2022; Russier *et al.*, 2012). For Lassa fever, RT-qPCR functions as a primary diagnostic tool, and the cycle threshold (Ct) value obtained from the diagnostic assay serves as an inversely proportional measure of the viral load (Choi *et al.*, 2018). In other viral diseases such as dengue and COVID-19, lower Ct values, which indicate greater viral loads, are associated with advanced, poorly controlled disease, and worse outcomes. In the case of dengue, poor outcomes are associated with high levels of viral load in the bloodstream, or viremia (Bhatt *et al.*, 2024).

Compared to other infections, predicting clinical outcomes in Lassa fever based on Ct values is still the most difficult to achieve. This case series study focuses on patients with Lassa fever who experienced adverse outcomes—severe complications or death despite presenting with higher Ct values, which, in most cases, are indicative of lower viral loads. Such scenarios deviate from the norm and pose a significant challenge to the current understanding of Lassa fever pathophysiology and, to a certain degree, the prognostic value of Ct values on its own. This paper intends to examine possible factors, especially the host immune response, together with the viral co-infection and disease comorbidity that may potentially explain the progression of the disease in these cases of advanced illness. Such analyses will contribute to refining the risk stratification and clinical management of patients with Lassa fever, ultimately improving patient outcomes. This aims to align with initiatives within the discipline to better understand the adverse events in the human body related to infectious diseases (Phipps *et al* ., 2024).

2.0 METHODOLOGY

Study Design and Setting: This paper describes a retrospective case series involving two laboratory-confirmed Lassa fever patients seen at the Infection Control and Research Centre (ICRC) of the Federal Medical Centre (FMC), Owo, Ondo State, Nigeria; a facility that provides care for patients with viral haemorrhagic fevers referred from southwestern Nigeria.

Case Identification and Diagnosis: Individuals reported on in this case series were confirmed as positive for Lassa virus (LASV) using reverse transcription polymerase chain reaction (RT-PCR) at the reference laboratory. The diagnosis was made on the basis of blood samples showing LASV RNA and other results including cycle threshold (Ct) values for the glycoprotein (G) and large (L) genes. A higher Ct value indicated lower viral load.

Data Collection: Data for this paper were clinical in nature and were captured in the medical records and case management forms at the ICRC. Clinical information obtained included sociodemographic information, clinical presentation, results of laboratory investigations, and details of therapeutic measures, complications, and outcomes. The haemogram, the renal and hepatic function tests, and the viral load as indicated by Ct values, were also analyzed.

Clinical Management: Both patients received standard Lassa fever management practice by the Nigerian Centre for Disease Control. Management included the administration of IV ribavirin (with adjustments for renal functions),

IV supportive fluid therapy, broad-spectrum antibiotics, blood transfusions, steroids, antipyretics, and blood transfusions as necessary. Patients with renal insufficiency received dialysis. All patients were cared for under isolation and control measures of the disease.

Ethical Considerations: This paper does not contain identifiable and potentially compromising information. The claims are grounded on anonymized data which was approved by the Federal Medical Centre Owo Institutional Review Board. Informed consent for publication was obtained by the hospital's ethical oversight unit.

3.0 RESULTS

Table 1. Summary of Clinical Findings and Progression – Case One

Day / Parameter	Clinical Events and Findings	Laboratory Results	Management / Outcome
Presentation (Day 0)	A 45-year-old male trader presented with 1-week history of fever and abdominal pain, 5-day history of coke-colored urine, and a 2-day history of cough and dyspnea. Fever was high-grade and continuous, associated with malaise, weakness, and anorexia. Abdominal pain was intermittent, burning, and localized to the epigastrium. No jaundice, hematochezia, or vomiting.	—	Provisional diagnosis: acute febrile illness likely viral hemorrhagic fever. Commenced on oxygen, IV fluids, antibiotics, antimalarial, PPI, and antipyretics.
Admission Findings (A/E)	Conscious but in respiratory distress, febrile (38.3°C), mildly pale and dehydrated, tachypneic (RR 32), tachycardic (PR 124), hypertensive (BP 160/90), oxygen saturation 80%.	—	—
Diagnosis	Lassa fever confirmed by PCR (Ct 32.81 and 31.37 on G and L genes).	—	Transferred to Isolation Centre (ICRC).
Day 1 (ICRC)	Febrile, pale, tachypneic (RR 38), confused (GCS 10/15), hypoxemic despite oxygen; oliguric with coke-colored urine.	—	IV ribavirin (renal dose), dexamethasone, co-amoxiclav, azithromycin, tranexamic acid, fluid restriction, close monitoring.
Day 2	Oliguria, hematuria, dyspnea, persistent fever.	PCV 25%; WBC 53,000; creatinine 1120 µmol/L; urea 24.1 mmol/L; AST 933 IU/L; ALT 218 IU/L; ALP 351 IU/L; albumin 24 g/L; metabolic acidosis.	Diagnosis: Lassa fever with ARDS, AKI, acute hepatitis, anemia, and hypoalbuminemia. Hemodialysis and blood transfusion done.
Day 3–4	Regained consciousness but remained oliguric and dyspneic.	Post-dialysis improvement in	Oral potassium commenced; antibiotics escalated to

		renal function; persistent leukocytosis; hypokalemia.	meropenem then levofloxacin; second dialysis performed.
Day 5–6	Persistent fever (38.6°C), dyspnea, oxygen dependence.	—	Third dialysis done. Developed generalized seizures during dialysis, treated with IV phenytoin.
Day 7	Deteriorated (GCS 8/15), persistent fever, oliguria, anemia.	PCV 24.3%; creatinine 414 µmol/L; urea 12.3 mmol/L; total bilirubin 141 µmol/L; repeat PCR positive (Ct ~36).	Continued on antipyretics, artesunate, broad-spectrum antibiotics; planned for another dialysis.
Day 8	Septic shock (BP 85/55, PR 150, T 40.7°C, GCS 5/15).	—	Despite IV fluids, dopamine, and supportive care, patient deteriorated and was certified dead.

Table 2. Summary of Clinical Findings and Progression – Case Two

Day / Parameter	Clinical Events and Findings	Laboratory Results	Management / Outcome
Presentation	37-year-old male truck driver with 2-week history of high-grade continuous fever, generalized weakness, headache, malaise, anorexia, and non-productive cough. Developed upper abdominal pain, vomiting, loose non-bloody stools, and 3-day history of coke-colored urine. One day before presentation: generalized tonic–clonic seizure.	—	Initially treated for malaria and typhoid without improvement. PCR confirmed Lassa fever (Ct 35.88/36.25).
On Admission (Day 0)	Lethargic (GCS 10/15), tachypneic (RR 32 cpm), oxygen saturation 97% on room air, afebrile, dehydrated, with moderate epigastric tenderness.	RBS 109 mg/dl.	Initial assessment: Lassa fever complicated by viral encephalopathy and possible AKI. Commenced on IV ribavirin, dexamethasone, antibiotics, tranexamic acid, omeprazole, fluids, and urine catheterization.
7 Hours Post-Admission	Developed seizure (RBS 148 mg/dl), semi-conscious (GCS 10/15), febrile (37.8°C), desaturated to 89–90%.	—	Oxygen via nasal prong, IV phenytoin and diazepam started.
Day 1–2	Recurrent seizures despite anticonvulsants; unconscious (GCS 8/15), hyperpyretic (40.1°C),	—	Oxygen therapy maintained; reviewed by anaesthesiology.

	desaturation requiring non-rebreather oxygen (15 L/min).		
Day 3	Persistent seizures.	PCV 38.2%; WBC 7,930/µL; platelets 267,000/µL; creatinine 671.2 µmol/L; urea 29.2 mmol/L; albumin 27.9 g/L; electrolytes normal.	Diagnosis revised to Lassa fever with AKI, ARDS, and hypoalbuminemia. Underwent hemodialysis.
Day 4	Progressive clinical decline.	—	Fifteen hours post-dialysis, developed agonal breathing. Despite resuscitation, patient was certified dead.

4.0 DISCUSSION

In Case One, table 1, A 45-year-old male trader presented to the Accident and Emergency (A/E) unit with a one-week history of fever, abdominal pain, and a five-day history of coke-colored urine, followed by a two-day history of cough and dyspnea. The fever was high-grade, continuous, partially relieved by antipyretics, and associated with malaise, weakness, and anorexia. Abdominal pain was intermittent, burning, and localized to the epigastrium, with associated dark stools but no jaundice, hematochezia, or vomiting. Coke-colored urine was accompanied by increased frequency, without dysuria, oedema, or puffiness. Cough was non-productive, with chest discomfort, and breathing difficulty manifested as tachypnea and shortness of breath. There were no neurological symptoms initially and no history of rodent exposure, funerals, or contact with suspected cases.

He was previously treated for malaria and sepsis at a private facility without improvement. He had no significant past medical or surgical history, was married, and did not consume alcohol or tobacco. At A/E, the patient was conscious but in respiratory distress, febrile (38.3°C), mildly pale and dehydrated, tachypneic (RR 32), tachycardic (PR 124), hypertensive (BP 160/90), with oxygen saturation of 80%. There was epigastric tenderness. A provisional diagnosis of acute febrile illness, likely viral hemorrhagic fever, was made. He was commenced on oxygen, IV fluids, antibiotics, antimalarial, PPI, and antipyretic. Laboratory investigations were sent, and Lassa fever PCR returned positive (Ct values ~32). He was transferred to the Isolation Centre (ICRC). On arrival at ICRC, he remained febrile, pale, tachypneic (RR 38), and confused (GCS 10/15), with persistent hypoxemia despite oxygen therapy, necessitating escalation to a non-rebreather mask (15 L/min). He was oliguric with coke-colored urine. Assessment of Lassa fever complicated by ARDS (acute respiratory distress syndrome) and suspected AKI (acute kidney injury) was made. Management included IV ribavirin (renal dose), dexamethasone, antibiotics (co-amoxiclav, azithromycin), tranexamic acid, fluid restriction, and close monitoring.

Day 2: Patient remained oliguric, hematuric, dyspneic, and febrile. Investigations showed moderate anemia (PCV 25%), marked leukocytosis (WBC 53,000), metabolic acidosis, elevated creatinine (1120 µmol/L) and urea (24.1 mmol/L), liver enzyme derangement (AST 933 IU/L, ALT 218 IU/L, ALP 351 IU/L), and hypoalbuminemia (24 g/L). A final diagnosis of Lassa fever with ARDS, AKI, acute hepatitis, anaemia, and hypoalbuminemia was made. He had hemodialysis and a blood transfusion.

Day 3–4: He regained consciousness but remained oliguric and dyspneic. Post-dialysis results showed partial improvement in renal function but persistent leukocytosis and hypokalemia, for which oral potassium was commenced. Antibiotics were escalated to meropenem and later levofloxacin. He had a second dialysis.

Day 5–6: Condition worsened with persistent fever (38.6°C), dyspnea, and oxygen dependence. He was assessed as having Lassa fever with severe sepsis, AKI, and ARDS. A third dialysis was performed. During dialysis, he developed generalized seizures, requiring IV phenytoin.

Day 7: He deteriorated with GCS 8/15, persistent fever, oliguria, and anemia. Post-dialysis labs revealed severe anemia (PCV 24.3%), creatinine 414 µmol/L, urea 12.3 mmol/L, and marked hyperbilirubinemia (T. bilirubin 141 µmol/L). Repeat PCR was still positive (Ct ~36). He was managed for Lassa fever with severe sepsis, AKI, and anemia, on antipyretics, artesunate, broad-spectrum antibiotics, and was planned for another dialysis.

Day 8: He developed septic shock (BP 85/55, PR 150, T 40.7°C, GCS 5/15). Despite IV fluids, dopamine, and aggressive supportive care, he further deteriorated and was certified dead.

Table 2 (case 2) showed that A 37-year-old male truck driver was transferred from the Accident and Emergency unit with confirmed Lassa fever. He had a two-week history of high-grade continuous fever, generalized weakness, headache, malaise, anorexia, and a distressing non-productive cough. He later developed upper abdominal pain, recurrent vomiting, loose non-bloody stools, and three days of passing coke-coloured urine. One day before presentation, he had a generalized tonic–clonic seizure. There was history of rodent exposure but no funeral attendance or contact with symptomatic individuals. He was initially treated for malaria and typhoid without improvement before testing positive for Lassa fever (Ct 35.88/36.25).

On admission, he was lethargic, GCS 10/15, tachypneic (RR 32cpm), saturating 97% on room air, afebrile, not pale or icteric, but dehydrated. Cardiovascular status was stable, and the abdomen showed moderate epigastric tenderness without organomegaly. Random blood sugar was 109 mg/dl. Initial assessment was Lassa fever complicated by viral encephalopathy with possible acute kidney injury. He was admitted, commenced on intravenous ribavirin, dexamethasone, antibiotics, tranexamic acid, omeprazole, fluids, and catheterization for urine monitoring. Seven hours into admission, he developed another seizure (RBS 148 mg/dl), became semi-conscious (GCS 10/15), febrile (37.8°C), and desaturated to 89–90%. He was started on oxygen via nasal prong, then phenytoin and diazepam. By the next day, he had recurrent seizures despite anticonvulsants, became unconscious (GCS 8/15), hyperpyretic (40.1°C), and desaturated further. He required escalation to a non-rebreather mask oxygen at 15 L/min, improving saturations to 94% after an anesthesiology review. On day three, seizures persisted. Investigations revealed PCV 38.2%, WBC 7,930/µL, and platelets 267,000/µL. Electrolytes were normal, but creatinine was markedly elevated (671.2 µmol/L), urea was 29.2mmol/L, and hypoalbuminemia (27.9 g/dL). Diagnosis was revised to Lassa fever complicated by acute kidney injury, ARDS, and hypoalbuminemia. He underwent hemodialysis but continued to have breakthrough seizures. Despite aggressive supportive management, his condition worsened. About fifteen hours post-dialysis, he developed agonal breathing. Resuscitation attempts failed, and he was certified dead on the fourth day of admission.

5.0 CONCLUSION

This report elaborates on the clinical course, the management, and the results of two confirmed cases of Lassa fever arising at a national referral centre. Prompt diagnosis using RT-PCR, the commencement of ribavirin therapy, and the provision of supportive care greatly contributed to the positive outcomes observed in the cases. The need to maintain a high index of clinical suspicion of Lassa fever in specific regions, broadening the clinical Lassa fever diagnostic and management horizon, and the prescribed treatment and prevention of nosocomial spread of the disease are all reinforced by the cases.

Recommendation

Timely ribavirin therapy is also critical; its effectiveness, though, declines markedly if started beyond six days of symptom onset. (Ogbaini-Emovon *et al.* .,2024). Therefore, prognosis should not rely solely on viral load, but on comprehensive clinical and immunological evaluation

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