

LASSA FEVER COMPLICATED BY ACUTE RESPIRATORY DISTRESS SYNDROME IN A HEALTHCARE WORKER: LESSON FROM A SUCCESSFUL MANAGEMENT EXPERIENCE AT FMC OWO

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Author's contributions

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, a viral hemorrhagic disease endemic to West Africa, remains a major public health concern, particularly when complicated by acute respiratory distress syndrome (ARDS). Pulmonary involvement is relatively uncommon, occurring in approximately 3–20% of hospitalized cases, yet it is associated with mortality rates as high as 70%. The pathologic features of Lassa fever-associated ARDS include interstitial pneumonitis, pulmonary hemorrhage, and diffuse alveolar damage. Despite advances in supportive care, survival from this severe complication remains rare, especially among healthcare workers. We present a case report of a 29-year-old female healthcare worker diagnosed with Lassa fever complicated by ARDS. The patient was managed at the Federal Medical Centre (FMC) Owo Isolation Centre, Ondo State, Nigeria. Clinical data were retrieved from inpatient records, and management included standard antiviral therapy with intravenous ribavirin alongside comprehensive critical care support, mechanical ventilation, and multidisciplinary coordination. This case represents a rare survival outcome in Lassa fever complicated by ARDS. The patient's illness progressed from mild febrile symptoms to severe respiratory failure requiring invasive mechanical ventilation. Her recovery was attributed to three key factors: early initiation of ribavirin therapy, aggressive and sustained critical care interventions, and prompt identification and management of secondary complications, including steroid-induced hyperglycemia, bacterial pneumonia, and electrolyte imbalance. Despite several episodes of clinical deterioration—including a transient cardiac arrest—the patient achieved full recovery following 26 days of hospitalization. Serial RT-PCR testing demonstrated progressive viral clearance, and she was discharged home in stable condition with appropriate follow-up care. This case highlights that survival from Lassa fever-associated ARDS, is achievable with timely diagnosis, early ribavirin administration, and advanced multidisciplinary care. It also

underscores the occupational risks faced by healthcare workers and reinforces the need for strict infection prevention and improved intensive care capacity in resource-limited settings.

Keywords: Lassa fever; acute respiratory distress syndrome; Healthcare worker; Critical care; Favourable outcome

INTRODUCTION

Lassa fever is a viral haemorrhagic illness caused by the Lassa virus, an Arenavirus that remains a major public health concern in West Africa due to its persistent endemicity and recurrent outbreaks (Asogun *et al.*, 2019; Yun & Walker, 2012). Although infection is traditionally linked to contact with *Mastomys natalensis* rodents or exposure to contaminated bodily fluids, emerging research indicates that other animals such as domestic species and reptiles may potentially serve as reservoirs, expanding the understanding of its epidemiology (Happi *et al.*, 2024; Kenmoe *et al.*, 2020). While most infected individuals experience mild or non-specific symptoms, about one in five patients progress to severe disease characterized by haemorrhage, neurologic complications, and multiorgan dysfunction, with mortality rates surpassing 30% in these cases (Asogun *et al.*, 2019; Ilesanmi *et al.*, 2023). Pulmonary involvement is considered less common but is a critical predictor of poor outcomes.

Respiratory failure—particularly acute respiratory distress syndrome (ARDS)—occurs in a concerning proportion of hospitalized patients and is associated with mortality rates of up to 70% (Ilesanmi *et al.*, 2023; Okokhere *et al.*, 2012). Pathological changes in the lungs, including diffuse alveolar damage and pulmonary oedema, contribute to rapid clinical deterioration and therapeutic challenges (Kenmoe *et al.*, 2020). Despite growing recognition of these complications, survivors of Lassa fever complicated by ARDS remain exceptionally rare, leaving significant gaps in evidence-based management (Okokhere *et al.*, 2012). This case report describes the successful clinical management of a healthcare worker who developed ARDS secondary to Lassa fever. It also underscores the importance of prompt antiviral therapy, appropriate ventilatory support, and coordinated multidisciplinary care while drawing attention to the elevated occupational risk among healthcare workers in resource-limited environments (Mba *et al.*, 2020). This paper studies one of the few recorded survivals of Lassa fever-associated ARDS in Nigeria and outlines the critical interventions responsible for recovery.

CASE PRESENTATION

Initial Presentation: A 29-year-old Yoruba female physician undergoing her compulsory internship at Federal Medical Centre (FMC) Owo, Nigeria, presented to the staff clinic with a 3-day history of headache and generalized body weakness. She was initially managed for malaria on an outpatient basis. Due to persistent symptoms, her blood was sent for viral hemorrhagic fever (VHF) screening, which returned positive for Lassa fever via reverse transcription polymerase chain reaction (RT-PCR) with CT values of 38.84, 29.66 on the G and L genes.

Clinical Course: At admission to the Infection Control and Research Centre (ICRC), she reported a 5-day history of generalized headache and malaise, with 3 days of non-bloody, non-bilious, non-projectile vomiting and associated epigastric pain and anorexia. Notably, she reported contact with a confirmed case (a deceased neonate) and a needle stick injury one week prior. There was no history of rodent infestation at home, and food was stored in rodent-proof containers. Her medical history was unremarkable. On examination, she was acutely ill-looking and lethargic, but afebrile (36.1°C), anicteric, not pale, and without peripheral edema. Vital signs were stable: pulse rate 82 bpm, blood pressure 110/70 mmHg, respiratory rate 20 cpm, and oxygen saturation (SpO₂) 100% on room air. Physical examination was unremarkable, apart from mild epigastric tenderness.

Complication and Management: She was commenced on intravenous (IV) Ribavirin 5 days into the acute illness per the Irrua regimen: 6.3 g stat, then 1.6 g daily for six days, followed by 800 mg daily for three days.

Empiric antibiotics (IV ceftriaxone 1 g 12 hourly and metronidazole 500 mg 8 hourly), IV fluids (alternating 500 mL normal saline and 5% dextrose), and anti-emetics (IV metoclopramide 10 mg 8 hourly) were initiated. Investigations revealed leukopenia (WBC 3160/μL) with lymphocytes (13.5%), neutrophils (83%), thrombocytopenia (90,000/μL), hyponatremia (Na⁺ 129 mmol/L), borderline potassium (K⁺ 3.5 mmol/L), and metabolic acidosis (HCO₃⁻ 17 mmol/L). BUN (4.3mmol/L) and creatinine (90umol/L) were normal. She then developed muscle stiffness, tongue protrusion,

and restlessness after 24hrs—features suggestive of acute dystonic reaction to metoclopramide. Both metronidazole and metoclopramide were discontinued. She was treated with oral trihexyphenidyl (5 mg daily × 5 days) and IV diazepam (10 mg stat), with resolution of symptoms. Her SpO₂ declined to 94% by day 4 of admission, prompting initiation of oxygen via nasal prongs at 3 L/min. An assessment of superimposed hospital-acquired pneumonia was made. Repeat electrolytes showed further hypokalemia (K⁺ 2.76 mmol/L). Antibiotic coverage was modified to IV co-amoxiclav, and oral azithromycin; IV dexamethasone and IVF 500ml Ringer's lactate alternating 5% dextrose 4 hourly was commenced. IV ceftriaxone was discontinued.

She became acutely dyspneic 24hrs later, with SpO₂ of 75% and oxygen via non-rebreathing mask (15 L/min) was commenced. She was reviewed by the anaesthetists. RR was 46 cpm, PR 109 bpm, and haemoglobin was 9.7 g/dL. ARDS was diagnosed, and chest X-ray requested. She was nebulized with salbutamol, commenced on non-invasive CPAP, and later intubated and ventilated using SIMV mode (FiO₂ 0.9, PEEP 8 cmH₂O, tidal volume 500 mL).

Chest X-ray done showed patchy opacities consistent with pneumonia; COVID-19 test was negative. She received fresh whole blood transfusion following a PCV of 30%. Post transfusion PCV was 28%. Persistent desaturation with bilateral coarse crepitation and reduced air entry on auscultation, with a respiratory rate of 56 cycle per minute led to escalation to IV meropenem and continuation of azithromycin after initial 72hrs of IV co-amoxiclav without significant improvement. An NG tube was inserted for enteral feeding. IV amino acids and IV morphine and paracetamol were added. Nutritional support included fortified meals and supplements (zinc, vitamin C, and multivitamins). The ventilator was switched to CPAP mode by the anaesthetist on day 10 of admission, as patient was stable and her chest was clearer clinically and lung zones on plain radiograph; at FiO₂ of 0.7.

Recovery Phase: The result of 10th day post-ribavirin PCR was still positive with CT values of 36.81 and 32.71 on G and L genes respectively. She developed persistent fever (38.2°C), hypertension (up to 180/110 mmHg), and tachycardia (PR 130–170 beat per minute), RBS of 12.8mmol/L and D-dimer was elevated (10 ng/mL). An assessment of steroid induced hyperglycemia, venous thromboembolism, and severely elevated BP in a previously normotensive patient was made. Endotracheal aspirate cultures grew *Staphylococcus aureus* sensitive to levofloxacin, ciprofloxacin and imipenem. IV levofloxacin was then added to her medications. Suspecting steroid-induced hyperglycemia, she was started on subcutaneous insulin, and oral dabigatran, carvedilol and natrilix were introduced. IV Ribavirin at 12.5 mg/kg daily was extended for 5 days. She was also transfused with 1 unit of fresh whole blood under diuretic cover.

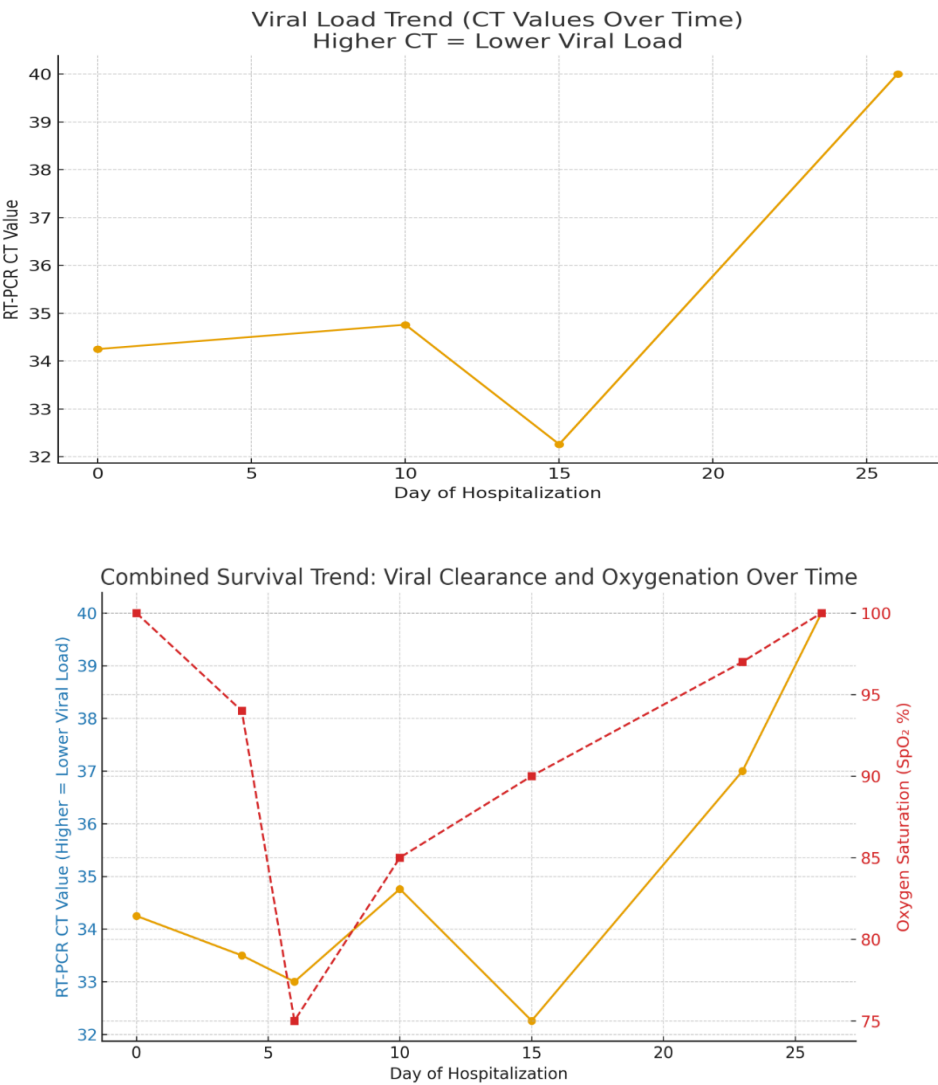
She experienced a cardiac arrest on day 12 but was successfully resuscitated with CPR and adrenaline. She was re-intubated and placed on mechanical ventilator by the anaesthetists. After clinical stabilization, she was extubated and placed back on CPAP. Electrolyte correction and insulin adjustments were continued. Insulin was discontinued on day 15 following normalization of fasting blood sugar (4.8mmol/L). Steroids were tapered. Oxygen therapy was de-escalated to nasal prongs (5 L/min), and antihypertensive therapy was adjusted. A repeat Lassa RT-PCR on day 15 remained positive with CT values of 32.92 on G gene and 31.60 on L gene, and IV Ribavirin was extended at 12.5 mg/kg for an additional 5 days per NCDC protocol and thus necessitating a central venous catheter as a result of difficult IV access. Persistent hyperglycemia required recommencement of insulin (14 IU tds pre-meal). Severe hypokalemia (K⁺ 2.4 mmol/L), on day 20 was corrected with IV potassium chloride. Post transfusion PCV was 29%. The patient showed significant clinical improvement, Astyfer capsule was commenced the following day, NG tube was removed, she was encouraged to ambulate. IV antibiotics were discontinued, while oral levofloxacin was commenced. She commenced chest physiotherapy and was weaned off oxygen support on day 23, resumed oral feeding, and gradually returned to baseline function. A repeat Lassa RT-PCR on day 26 was negative. FBS was 7.9 mmol/L. she was discharged by the endocrinologist on subcutaneous insulin for subsequent follow up. Tab dabigatran was discontinued and she was placed on tab clopidogrel. Repeat D-dimer was 2.43ng/L. She was discharged home and scheduled for follow up at the various clinics.

OUTCOME SUMMARY

Following admission, the patient's clinical course demonstrated rapid progression from mild symptoms to severe respiratory compromise. Initial laboratory investigations revealed leukopenia, thrombocytopenia, mild metabolic acidosis, and electrolyte imbalance. Despite commencement of antiviral therapy and supportive treatment, her

respiratory status deteriorated significantly by day 4 of hospitalization, prompting escalation from oxygen therapy via nasal prongs to non-invasive ventilation and subsequently invasive mechanical ventilation for ARDS management.

Serial chest examinations and radiographic findings confirmed diffuse bilateral infiltrates consistent with pneumonia and ARDS. During mechanical ventilation, she developed a multidrug-resistant *Staphylococcus aureus* ventilator-associated pneumonia, which required antibiotic modification based on culture sensitivity. Additional complications included steroid-induced hyperglycaemia, persistent hypokalaemia, hypertensive crisis, elevated D-dimer suggestive of thromboembolic risk, and a transient cardiac arrest—each managed promptly with favourable response. Over the following days, gradual clinical improvement was observed. Oxygenation stabilized, enabling successful transition from invasive ventilation to CPAP and eventual discontinuation of supplemental oxygen by day 23. Serial RT-PCR for Lassa virus demonstrated declining viral load, converting to negative on day 26. Functional recovery progressed steadily, including tolerance of enteral nutrition, mobilization, and normalization of laboratory parameters. She was discharged home in stable condition after 26 days of hospitalization, with appropriate outpatient follow-up arranged for metabolic monitoring and pulmonary rehabilitation.



The combined trend demonstrates a strong inverse relationship between viral load and oxygenation status during the patient’s hospitalization. In the early phase of illness, CT values were relatively low, indicating high viral replication, while SpO₂ progressively declined, and ultimately requiring invasive mechanical ventilation. By day 10, a modest improvement in oxygenation coincided with a rising CT value, suggesting partial virologic response to ribavirin therapy.

The lowest oxygen saturation and most severe respiratory compromise occurred around day 6, which aligned with ongoing viral activity and the development of ARDS. Following escalation of ventilatory support and targeted management of complications—including ventilator-associated pneumonia—SpO₂ steadily improved. By day 26, CT values reached the threshold considered negative for viral detection, demonstrating complete viral clearance. This coincided with the return of normal oxygenation, weaning from respiratory support, and clinical stabilization. Overall, the trend illustrates that clinical recovery closely paralleled virologic suppression, reinforcing the benefit of timely antiviral administration and robust critical care interventions in improving survival outcomes in Lassa fever complicated by ARDS.

DISCUSSION

Healthcare workers remain disproportionately vulnerable to adverse outcomes from Lassa fever due to their frequent exposure to infectious bodily fluids, insufficient access to personal protective equipment (PPE), and low initial suspicion for viral hemorrhagic fevers in clinical settings according to (Mba *et al.*, 2020). These challenges contribute to increased occupational transmission and reinforce the urgency of strengthening infection prevention and control practices in endemic areas (Mba *et al.*, 2020). This case highlights an uncommon but successful recovery from Lassa fever complicated by ARDS—an entity associated with mortality rates of up to 70% among hospitalized patients (Okokhere *et al.*, 2012). Three major clinical actions appeared pivotal to the patient's favorable outcome: early initiation of ribavirin, the use of advanced critical care interventions, and timely management of secondary complications.

Ribavirin remains and it's proven to be the only widely available antiviral therapy proven to improve survival in Lassa fever and is most beneficial when administered within the first six days of illness onset (Mba *et al.*, 2020). In this case, ribavirin was initiated early; however, the progression to ARDS indicates that antiviral therapy alone may not be sufficient in cases with pronounced pulmonary involvement, supporting previous observations (Yun & Walker, 2012). Further research into adjunctive therapies—such as monoclonal antibodies and host immunomodulators—may offer additional benefit in severe presentations.

Aggressive critical care support also played a crucial role. The patient required escalation from non-invasive respiratory support to invasive mechanical ventilation with lung-protective strategies. Although evidence guiding ventilation approaches in Lassa-associated ARDS is still limited, interventions that minimize barotrauma and reduce alveolar stress likely contributed to stabilization (Asogun *et al.*, 2019; Ilesanmi *et al.*, 2023). Additionally, hospital-acquired multidrug-resistant bacterial pneumonia complicated her respiratory failure, underscoring the need for judicious and targeted antibiotic therapy during prolonged intensive care unit admission.

Furthermore, the multidisciplinary management of secondary complications—including suspected thromboembolism, steroid-induced hyperglycemia, electrolyte derangements, and a brief cardiac arrest—was essential to recovery. Continuous physiological monitoring enabled early detection and appropriate treatment adjustments throughout her course of illness. Together, these factors demonstrate that survival from Lassa fever complicated by ARDS is possible when rapid diagnosis, timely ribavirin administration, and high-quality critical care are available. However, the case also reinforces the significant occupational hazards faced by frontline healthcare professionals in resource-limited settings, emphasizing the need for stronger PPE availability, adherence to infection control protocols, and improved critical care capacity across endemic regions.

Finally, this case brings to fore the fact that nosocomial transmission to healthcare workers remains a preventable risk; therefore strict PPE protocols and ribavirin prophylaxis for exposures should be enforced. Endemic regions must also prioritize developing ICU capacity for Lassa fever care given the high mortality associated with pulmonary complications. Finally, further studies are needed to explore the role of adjuvant therapies and biomarkers for ARDS prediction. The limitations were limited autopsy data to confirm ARDS histopathologically and the inability to assess long-term pulmonary sequelae post-recovery.

CONCLUSION

This case demonstrates a rare survival from Lassa fever-associated ARDS, underscoring the importance of early critical care intervention, vigilant management of secondary infections, and multidisciplinary coordination. Healthcare workers in endemic regions remain at high risk, emphasizing the need for stringent infection control measures. However, this case provides valuable insight into the possibility of successful outcomes in Lassa fever complicated by

ARDS—an otherwise often fatal manifestation. The patient's recovery underscores the essential importance of early antiviral therapy, access to robust critical care services, and coordinated multidisciplinary management in addressing both primary disease progression and secondary complications. Healthcare workers in Lassa fever–endemic settings remain at heightened occupational risk, highlighting the urgent need to enhance infection prevention and control systems, ensure adequate PPE availability, and continue investment in intensive care capacity. Ongoing research is further required to establish optimal treatment strategies for pulmonary complications and to explore innovative adjunctive therapies that may improve survival in severe Lassa fever cases.

RECOMMENDATIONS

To reduce the burden of severe Lassa fever outcomes and improve survival among healthcare workers in endemic regions, the following measures are recommended:

Provision of Personal Prevention systems: Hospitals should ensure consistent availability and appropriate use of personal protective equipment, along with frequent staff training to maintain a high index of suspicion for Lassa fever in clinical settings.

Improve early diagnostic capacity and treatment initiation: Rapid diagnostic testing and prompt administration of ribavirin are essential to improving prognosis and preventing progression to severe disease, especially for frontline workers.

Establish standardized clinical guidelines for Lassa-associated ARDS: Evidence-based protocols for ventilation strategies, fluid balance, and adjuvant therapies are needed to optimize clinical decision-making in severe pulmonary involvement.

Promote surveillance, research, and clinical trials: Further investigation into immunomodulators, monoclonal antibodies, and biomarkers of disease severity will help identify additional treatment options and enable prediction of ARDS risk.

Implement post-exposure prophylaxis policies for healthcare workers: Structured protocols for managing occupational exposure—including ribavirin prophylaxis and follow-up—can minimize preventable infections and reinforce workplace safety.

Limitations:

This case report reflects the unique clinical course of a single patient and therefore may not be generalizable to all individuals with Lassa fever–associated ARDS. Confirmation of ARDS pathology was limited by the absence of advanced imaging and histopathological evaluation, which were unavailable due to biosafety and resource constraints. Additionally, long-term pulmonary function and quality-of-life outcomes could not be fully assessed beyond the acute recovery phase. These limitations highlight the difficulties in conducting comprehensive research during outbreaks of viral hemorrhagic fevers in resource-limited settings.

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Conflict of Interest Declaration:

The authors declare no conflicts of interest in this study.

Author Contributions

The authors led clinical data acquisition, conceptualization, and manuscript drafting. OOA and EJF contributed to patient management, literature review, and manuscript revision. AAS, OO, and LAA provided specialist input in critical care, clinical supervision, and review of the final manuscript. All authors approved the final version of the manuscript and agree to be accountable for its content.

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