Case report

A Case Report of Lassa Fever Complicated by Acute Respiratory Distress Syndrome in a Healthcare Worker with a Favourable Clinical Outcome

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ABSTRACT

Background: Lassa fever, a viral haemorrhagic fever endemic to West Africa, carries a high mortality rate, particularly when complicated by acute respiratory distress syndrome (ARDS). Pulmonary involvement, though rare in about 3-20% of cases, is associated with a case fatality rate of up to 70%, compared to 38.2% in patients without respiratory complications. Histopathological findings in Lassa-associated ARDS include interstitial pneumonitis, pulmonary haemorrhage, and diffuse alveolar damage.

Methods: This study is a case report of Lassa fever complicated by acute respiratory distress syndrome in a healthcare worker that was managed at the Federal Medical Centre, Owo (FMCO) isolation centre with a favourable outcome.

Results: This case represents a rare favourable outcome in Lassa fever complicated by ARDS. The patient's survival likely resulted from three key factors namely: early ribavirin administration, aggressive critical care support, and prompt management of secondary complications. While our patient received ribavirin early, her progression to ARDS suggests that antiviral therapy alone may be insufficient in severe pulmonary involvement; a finding consistent with prior reports.[7] In addition, aggressive critical care support was key as the patient's hypoxemia required escalating support from non-invasive ventilation to invasive mechanical ventilation. Furthermore, prompt management of the secondary complications through multidisciplinary approach was vital to this patient's survival.

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.

Keywords: *Lassa fever; Pulmonary complications; Favourable outcome; Healthcare worker*

Introduction

Lassa fever, caused by the Lassa virus (an Arenavirus), is a zoonotic disease endemic to West Africa, with primary transmission occurring through exposure to Mastomys natalensis rodents or infected bodily fluids.[1] Recent evidence suggests potential non-rodent reservoirs, including lizards and domestic animals, though their epidemiological role remains under investigation.[2] The disease manifests as a mild febrile illness in 80% of cases, but severe presentations such as haemorrhagic shock, encephalopathy, and multi-organ failure, carries mortality rates exceeding 30%.3

Pulmonary complications, though uncommon, significantly worsen prognosis.[4] ARDS develops in 3-20% of hospitalized patients with Lassa fever and is associated with a mortality rate of 70%.4 Pathological mechanisms include diffuse alveolar damage, pulmonary oedema, and secondary bacterial pneumonia.[5] Few documented survivors of Lassa-associated ARDS exist, thus making management strategies poorly defined.

This report details a rare case of a healthcare worker who survived Lassa fever complicated by ARDS, highlighting the critical roles of early ribavirin, ventilator strategies, and multidisciplinary care. The case also reinforces the occupational risks faced by medical personnel in resource-limited settings.

Case 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.

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 (SpO□) 100% on room air. Physical examination was unremarkable, apart from mild epigastric tenderness.

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 (HCO□□ 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 SpO□ 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 ringers lactacte alternating 5% dextrose 4 hourly was commenced. IV ceftriaxone was discontinued.

She became acutely dyspneic 24hrs later, with SpO□ 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 (FiO□ 0.9, PEEP 8 cmH□O, 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 cpm 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. 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 bpm), 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 as well. She 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 7.9mmol/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 finally discharged home and scheduled for follow up at the various clinics.

Discussion

Healthcare workers are considered at risk of developing severe Lassa fever outcomes.[5,6] In fact, a high prevalence of Lassa fever has been recorded among healthcare workers as a result of low index of suspicion of Lassa fever, as well as inadequate availability of personal protective equipment for use.[6] All these make compliance to infection prevention and control measures difficult among healthcare workers.[6]

This case represents a rare favourable outcome in Lassa fever complicated by ARDS, a condition typically associated with 70% mortality.[4] The patient's survival likely resulted from three key factors namely: early ribavirin administration, aggressive critical care support, and prompt management of secondary complications.

On timely ribavirin therapy; ribavirin, being the only proven therapy for Lassa fever is considered most effective when initiated within the first 6 days of illness.[6] While our patient received ribavirin early, her progression to ARDS suggests that antiviral therapy alone may be insufficient in severe pulmonary involvement; a finding consistent with prior reports.[7]Adjunctive therapies, such as monoclonal antibodies, interferons, and other immunomodulators warrant exploration in future cases.

In addition, aggressive critical care support was key, as the patient's hypoxemia required escalating support from non-invasive ventilation to invasive mechanical ventilation. Lung-protective strategies such as low tidal volume and high PEEP, likely mitigated further alveolar injury, though data on optimal ventilation in Lassa-associated ARDS remain scarce. The development of MDR Staphylococcus aureus pneumonia further complicated her course, emphasizing the need for broad- spectrum antibiotics in prolonged ICU stays.

Furthermore, prompt management of the secondary complications through multidisciplinary approach was vital to this patient's survival. The suspected thromboembolism, a complication of viral haemorrhagic fevers, necessitated prophylactic enoxaparin, though confirmatory imaging was unavailable. The steroid-induced hyperglycaemia experienced, required insulin titration, reflecting the metabolic derangements seen in critical illness. The brief cardiac arrest suffered, likely due to metabolic acidosis, was rapidly reversed, highlighting the importance of continuous hemodynamic monitoring.

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.

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