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Divergent pathways of severe Lassa fever: Vascular leak and hyperinflammation versus neurological disease, recommendations for therapeutic countermeasure from the pathogenesis study

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Introduction

Lassa fever (LF) is a viral haemorrhagic fever (VHF) prevalent in West Africa with case fatalities rates in hospitalised patients ranging from 20-30 %. Previous studies have demonstrated acute kidney injury and encephalopathy as key symptoms associated with fatal outcome. Yet to date the underlying pathophysiology is poorly understood hampering the development of adequate medical countermeasures (MCM).

Methods

From January 2022 until December 2023, we recruited a prospective observational cohort of 329 adult Lassa fever patients at Irrua Specialist Teaching Hospital in Nigeria. Bidaily study visit included assessment of clinical exams and blood sampling. Patients were classified according to common outcome definitions. To decipher the pathogenesis of AKI and encephalopathy, patients with such diagnosis were recruited into sub-cohorts with additional focused assessments such as ultrasound, urinalysis, neurological exams and electroencephalography (EEG).

Results

The mean age of the cohort was 36.8 years and 42.7 % (137/321) were female. All patients were treated with Ribavirin and 48% also with Dexamethasone. The most common complications were acute kidney injury (AKI; 28% 96/342), hepatitis (23%, 79/342), and meningitis or focal neurological defect (8.5%, 29/342). AKI was mainly associated with hyperinflammation and a vascular leak syndrome. Fatal cases, compared with survivors, showed marked inflammation (CRP: 33 [10 – 71] vs. 98 [64 – 162], WBC: 6.7 [5.1 – 8.8] vs. 13.4 [8.9 – 25.2]) and, to a lesser extent, coagulopathy (INR: 1.07 [1.03 – 1.13] vs. 1.20 [1.05 – 1.28]). In neurologically impaired patients, distinct EEG patterns indicated viral meningitis.

Conclusion

Fatal cases exhibited a multi-organ failure syndrome hallmarked by hepatitis and AKI. This syndrome was associated with vascular leak and inflammation, rather than severe haemorrhage. A secondary phenotype (approx. 8.5%, 29/342) of patients had primarily neurological disease. We recommend that therapeutic approaches also target vascular leak and inflammation and address neuroinvasive infection.