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Original Research Article

CLINICAL CHARACTERISTICS AND RENAL OUTCOMES OF LASSA FEVER IN PATIENTS WITH SICKLE CELL DISEASE IN ENDEMIC REGION OF NIGERIA

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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 (LF) is a viral haemorrhagic disease endemic to West Africa and is associated with significant morbidity and mortality, particularly when complicated by acute kidney injury (AKI). Sickle cell disease (SCD), characterized by chronic hemolysis and endothelial dysfunction, may predispose affected individuals to more severe manifestations of LF; however, the clinical interaction between these conditions remains poorly described. We reported a case of severe Lassa fever complicated by rapidly progressive AKI in a 16-year-old female with homozygous sickle cell disease (HbSS). Clinical features, laboratory parameters including Lassa virus PCR cycle threshold values, therapeutic interventions, and outcomes were retrospectively reviewed. The patient presented with high-grade fever, respiratory symptoms, hemoglobinuria, electrolyte derangements, marked leukocytosis, and worsening renal function. Lassa virus PCR confirmed active infection with low cycle threshold values. Despite aggressive supportive care, renal function deteriorated, necessitating multiple sessions of hemodialysis. Management included renal-adjusted intravenous ribavirin, blood transfusions, correction of metabolic abnormalities, and coordinated multidisciplinary care. Viral clearance was achieved by day 15, followed by gradual clinical recovery. This case highlights the synergistic severity of Lassa fever and sickle cell disease, particularly in precipitating hemolysis and acute kidney injury. Early diagnosis, prompt initiation of antiviral therapy, access to renal replacement therapy, and multidisciplinary management are

critical to improving outcomes in this high-risk population. Further studies are needed to define disease patterns, optimize antiviral dosing, and develop tailored management guidelines for patients with sickle cell disease in Lassa fever–endemic regions.

Keywords: Lassa fever; Sickle Cell Disease; Acute Kidney Injury; Hemolysis; Viral Haemorrhagic Fever.

INTRODUCTION

Lassa fever (LF) is a zoonotic viral haemorrhagic disease endemic to West Africa and remains a major cause of morbidity and mortality, particularly when complicated by multiorgan dysfunction such as acute kidney injury (AKI) (Asogun *et al.,* 2019; Happi *et al.,* 2019; Okokhere *et al.,* 2018). The Lassa virus exhibits tropism for endothelial and renal tissues, resulting in capillary leak, electrolyte derangements, and progressive multiorgan involvement, which significantly worsens clinical outcomes (Happi *et al.,* 2019; Uppala *et al.,* 2025). Mortality among hospitalized patients ranges from 15% to 20% but increases substantially in the presence of complications such as shock, pulmonary edema, and AKI (Asogun *et al.,* 2019; Happi *et al.,* 2019).

Sickle cell disease (SCD), a prevalent hemoglobinopathy in West Africa, is characterized by chronic hemolysis, endothelial dysfunction, immune dysregulation, and a heightened susceptibility to severe infections and renal complications (Rees *et al.,* 2010; Nath & Hebbel, 2015; Tebbi, 2022).

These pathophysiological features overlap with mechanisms implicated in severe LF, suggesting a potential for synergistic disease severity when both conditions coexist. However, despite this biological plausibility, the clinical interaction between LF and SCD remains poorly characterized in the literature. Reports describing the coexistence of LF and SCD are scarce. Available evidence is limited to a small number of recent case reports from endemic regions in Nigeria, including cases from Alex Ekwueme Federal University Teaching Hospital, Abakaliki, and Irrua Specialist Teaching Hospital, which document severe disease courses marked by profound hemolysis, high transfusion requirements, and dialysis-dependent AKI (Ugwu *et al.,* 2023; Ojor Ileli *et al.,* 2025). These observations suggest that SCD may predispose patients with LF to more aggressive clinical trajectories and poorer outcomes, yet systematic data are lacking.

In this context, we report a severe case of Lassa fever complicated by acute kidney injury in a patient with underlying sickle cell disease. This case provides an opportunity to further elucidate the synergistic effects of LF and SCD, particularly with respect to hemolysis, renal involvement, and the early need for renal replacement therapy. By highlighting these challenges, this report underscores the importance of heightened clinical suspicion, early multidisciplinary intervention, and the urgent need for targeted research to inform evidence-based management guidelines for this high-risk population in Lassa fever–endemic settings.

MATERIALS AND METHODS

Study Design:

This was a retrospective case report conducted at a tertiary healthcare facility in Nigeria, documenting the clinical course and management of severe Lassa fever complicated by acute kidney injury in a patient with sickle cell disease, using routinely collected clinical data.

Case Management and Clinical Assessment:

The patient underwent daily clinical assessments including vital signs, fluid balance monitoring, and evaluation for complications such as hemolysis, respiratory distress, and electrolyte disturbances. Multidisciplinary input was obtained from infectious disease specialists, paediatric nephrologist, paediatric hematologist, and general pediatricians.

Laboratory and Diagnostic Investigations:

Laboratory evaluation included complete blood count, renal and liver function tests, electrolytes, urinalysis, and Lassa virus PCR. PCR cycle threshold (CT) values were used to monitor viral load dynamics.

Data Collection:
Clinical, laboratory, and treatment data were retrospectively documented from admission through discharge, including interventions such as ribavirin administration, transfusions, dialysis sessions, and supportive therapy.

Ethical Considerations:
This study was conducted as a single retrospective review of routinely collected clinical data. Patient confidentiality was preserved by removing all personal identifiers and reporting aggregated findings. The hospital management and infection control and research centre approved data use for research purposes, and all patient information was anonymized. Additionally, patient consent was obtained. Formal ethical approval was waived in line with institutional and national guidelines for retrospective descriptive studies.

CLINICAL PRESENTATION
History:
The patient is a 16-year-old female with known sickle cell anemia (HbSS) diagnosed at 3 years of age and has been attending Haematology clinic in the last 12 years, regular on routine drugs but not on Hydroxyurea, she presented to the Infection and control Centre (ICRC) following a confirmed positive Lassa fever test obtained at the facility while on admission at the Children Emergency Room (CHER). She reported a four-day history of high-grade, continuous fever with chills and rigors. She also developed cough, central chest pain, and difficulty breathing; no associated orthopnea or paroxysmal nocturnal dyspnea. She noted a three-day history of passing dark ("coke-colored") urine, associated with reduced urinary output, epigastric abdominal pain, and nausea. There was no history of bleeding from any other orifice. Rodents were reportedly present in her home, but no contact with sick persons or recent travel. She received initial care at the Children's Emergency Room (CHER) for a likely hemolytic crisis and possible acute chest syndrome. She was transfused with one unit of Hb AA genotype packed red blood cells due to suspected LF, she was not exchanged. She was then commenced on IV ribavirin, ceftriaxone, omeprazole, and metronidazole. Positive Lassa PCR prompted transfer to ICRC for specialist care. She was only on folic acid and other routine drugs prior, with no known drug allergies.

Examination:
Findings revealed respiratory distress, pallor, facial swelling, epigastric tenderness, and SpO_2 of 95–98% on 5 L/min intranasal oxygen.

INVESTIGATIONS
Baseline investigations: Lassa PCR positive (CT 24.95 / 25.01); FBC: WBC 54,820/µL, PCV 26.1%, PLT 383,000/µL; electrolytes: Na-127 mmol/L, K-3.1–3.5 mmol/L, TCO_2-17–18 mmol/L; renal: Cr-788 µmol/L, BUN-25.3mmol/L; liver: ALP-4,631 IU/L, Albumin-34 g/L. Subsequent investigations as detailed in Table 1 below showed fluctuating PCV, persistent hematuria, worsening renal function, recurring hypokalemia, and gradually improving liver enzymes. Repeat Lassa PCR on Day 15 was negative, confirming viral clearance.

Table 1: Laboratory results timeline (day – intervention – outcome)

DAY	KEY LAB FINDINGS	INTERVENTION	OUTCOME
1 (Children Emergency Room) (CHER)	PCV 19%, PCR for Lassa fever positive CT value 24.95(GPC) and 25.01(L)	Transfusion with 1 unit of blood. Commencement of IV ribavirin, ceftriaxone, omeprazole	PCV 26% Transferred patient to the Infection Control and Research Centre (ICRC), after initial few hours admission at the Children Emergency Room (CHER)

2 (Infection Control and Research Centre) (ICRC)	PCV-26.1%, WBC 54,820/µL, PLT 383,000/ µL, Na-127-mmol/L, K-3.1mmol/L, TCO_2-17mmol/L; Cr-788µmol/L, BUN-25.3mmol/L; AST-94IU/L, ALT-86IU/L, ALP-463IU/L, Albumin-34g/L	Parenteral medications were sustained; IV Ribavirin was reduced to 50% of current dose (Renal dose), scheduled for the first session of haemodialysis owing to abnormal biochemical parameters	Renal support initiated
4	CRE-989µmol/L, BUN-31.6mmol/L Na-128mmol/L, K-3.1mmol/L, Tco2-18mmol/l.	Scheduled for second session of hemodialysis owing to abnormal biochemical parameters still	Status quo
5	-	Had second session of hemodialysis	Status quo
6	FBC revealed WBC-37,020/µL PCV-29.3%. E/U/Cr revealed K-2.7mmol/L, BUN-12.4mmol/L, CRE-604µmol/L.	Consulted with the pediatric nephrologist	Reduction IV ciprofloxacin 200mg to 100mg 12hourly commenced on chest physiotherapy, and oral Slow K at 40mmol over 24hours.
8	Tco2-12mmol/l, BUN-18.2mmol/L, CRE-981µmol/L.	Had 3rd session of hemodialysis due to non-corrected biochemical parameters	Status quo
9	CRE-1011µmol/L, BUN-20.4mmol/L, Na-121mmol/L, K-2.7mmol/L, Tco2-10mmol/l, PCV-30.5%, WBC-33,020/µL	Had 4th session of hemodialysis	Status quo
10	Na-119mmol/L, K-2.4mmol/L, HCO3-22mmol/L, BUN-11.2mmol/L, CRE-491µmol/L. The result of her repeat Lassa virus PCR-(GPC) of 39.62.	10mmol of KCL was added to 500mls normal saline 12hourly	Extension of Ribavirin via tabs, discontinuation of potassium correction
14	PCV-23%	One unit of blood transfused	Post-transfusion PCV improved to 26%
15	Urea-21.1mmol/L, CRE-561µmol/L K-3.5mmol/L, Na-123mmol/L. PCR Negative for Lassa fever	Continued calcium lactate and rehydration with intravenous fluid Ringer's lactate; in addition to daily weighing	Serum Potassium level improved, Transferred patient to the pediatric ward.

TREATMENT

The treatment as shown in Table 2 below, included: renal-adjusted IV ribavirin, IV ceftriaxone, IV ciprofloxacin, IV metronidazole, IV omeprazole, IV tranexamic acid, IV furosemide, IV $NaHCO_3$, IV salt-poor albumin, IV fluids, oral spironolactone, potassium and calcium supplementation, oxygen therapy, chest physiotherapy, blood transfusion, and four hemodialysis sessions.

Table 2: Treatment timeline (day – intervention – outcome)

DAY	CLINICAL FINDINGS	INTERVENTION	OUTCOME
1	Respiratory distress; pale; facial swelling; epigastric tenderness; SpO_2 95–98% on 5L INO_2. Baseline labs: WBC-54,820/µL; PCV-26.1%; PLT-383000/ µL; Na-127 mmol/L; BUN-25.3 mmol/L; Cr-788 µmol/L; AST-94IU/L, ALT-86IU/L, ALP-463IU/L; TCO_2-17; Alb-34g/L, dark-colored urine.	Continued IV ribavirin (renal dose 50%); ceftriaxone; metronidazole (due to leucocytosis and persistent fever); omeprazole; started IVF 5% dextrose in Ringer's Lactate (150% maintenance). Counseled patient & caregiver. Baseline labs done.	Stabilized on oxygen; symptoms persisted.
2	Fever 38.4°C; persistent dark-colored urine; cough with bloody sputum; Urine output (UO) <1 mL/kg/hr; SpO_2-99% on INO_2.	Started IV tranexamic acid 250 mg 8-hourly; 1st hemodialysis session (due to abnormal biochemical parameters).	Persistent symptoms; remained hemodynamically stable.
3	Fever 38.3°C; respiratory distress ongoing; Urine output (UO)-0.73 mL/kg/hr. post-dialysis labs: Cr-989 µmol/L; BUN-31.6mmol/L; Na-128 mmol/L; K-3.1mmol/L; TCO_2-18 mmol/L.	Continued prior treatments.	Mild biochemical improvement; symptoms persisted.
4	Mild epigastric tenderness; persistent fever.	Discontinued IV omeprazole; switched antibiotics from IV ceftriaxone to IV ciprofloxacin 200 mg 12-hourly; started ACT (3 days).	Slight abdominal relief; fever persisted.
5	Symptoms persisted; worsening facial/periorbital edema, bilateral pitting edema, mild ascites.	2nd hemodialysis session (due to non-corrected biochemical parameters); continued medications.	Worsening fluid retention.
6	Persistent epigastric pain; dyspnea; UO-0.34 mL/kg/hr. Labs: WBC-37,020/µL; PCV-29.3%; K-2.7mmol/L; BUN-12.4 mmol/L; Cr-604µmol/L.	Reduced IVF to 1 L/day; reduced IV ciprofloxacin (100 mg 12-hourly due to renal function); restarted IV omeprazole 20 mg daily; chest physiotherapy started; oral Kcl 40 mmol/day.	Slight biochemical improvement; persistent oliguria.
7	Afebrile (37.2°C); bradycardia (PR-55 bpm); UO-0.17 mL/kg/hr. Labs: TCO_2-12 mmol/L; BUN-18.2mmol/L; Cr-981µmol/L. ECG-normal.	Continued supportive care.	Bradycardia monitored; renal parameters worsened.
8	Persistent anasarca; respiratory symptoms improving.	3rd hemodialysis session (biochemical parameters not yet corrected).	Respiratory distress resolving; renal status still poor.

9	Respiratory distress subsiding; fever resolved; persistent anasarca; UO-0.2 mL/kg/hr.	Started IV $NaHCO_3$ 2 mmol/kg/day; IV frusemide 2 mg/kg 12-hourly (48 hrs); spironolactone 25 mg 12-hourly; discontinued urethral catheter; reduced oxygen to 1.5 L/min.	Maintained SpO_2 99–100%; persistent oliguria & edema.
10	Hypothermia (34°C); desaturation (SpO_2 88–91%); UO-0.2 mL/kg/hr; worsening edema.	Warm blanket; oxygen increased to 4 L/min**;** 4th hemodialysis session**;** given IV salt-poor albumin; adjusted oxygen to 2 L/min, then concentrator 1 L/min.	SpO_2 improved to 97–100%; Creatinine improved to a post-dialysis value of (491); Post-ribavirin PCR now reduced to single gene positive (GPC 39.62).
11	(No major new symptoms documented)	Continued supportive therapy.	Stable clinical condition
12	No new complaints; UO improved to 0.75 mL/kg/hr, serum calcium-1.6mmol/L	Started oral Astymin; weaned off oxygen; discontinued tranexamic acid; continued oral KCl; started oral calcium lactate 650 mg 8-hourly.	Clinical improvement.
13	No new complaints	Started oral Astymin; weaned off oxygen; continued calcium lactate.	Clinical improvement.
14	Complained of body weakness, dizziness and easy fatigability	Urgent PCV requested was 23%, Transfused with 1 unit of blood	Clinical improvement
15	UO improved to 1.6 mL/kg/hr; persistent oedema; labs: BUN-21.1 mmol/L; Cr-561μmol/L; K-3.5 mmol/L; Na-123 mmol/L, ALT-16IU/L, AST-10IU/L, ALP-79IU/L, ALB-23g/L	Continued supportive therapy. Lassa PCR Negative**.**	Significant improvement; transferred to pediatric ward for continuation of care.

DISCUSSION

Lassa fever (LF) in individuals with homozygous sickle cell disease (SCD) is rarely reported, resulting in limited evidence to guide clinical management. Both conditions independently promote systemic inflammation, endothelial injury, and multiorgan dysfunction; their coexistence is therefore biologically plausible to produce amplified disease severity (Ojor Ileli *et al.,* 2025; Ugwu *et al.,* 2023). In SCD, chronic hemolysis and baseline inflammation increase vulnerability to intravascular hemolysis during systemic infections, with downstream risks of acute tubular injury and acute kidney injury (AKI) irrespective of viral characteristics (Tebbi, 2022; Yusuf *et al.,* 2017; Nath & Hebbel, 2015). Viral infections, including arenaviruses, are known to exacerbate oxidative stress and erythrocyte membrane instability, potentially precipitating hemolytic crises (Tebbi, 2022; Guasch *et al.,* 2006). In this patient, profound hemolysis manifested by rapid hematocrit decline, transfusion requirement, and persistent coke-colored urine likely reflected overlapping contributions from sickle-related hemolysis and LF-associated hepatocellular dysfunction—both recognized features of severe LF (Ojor Ileli *et al.,* 2025; Ugwu *et al.,* 2023). The marked leukocytosis (>54 × 10^9/L), atypical for LF which usually presents with leukopenia, likely represents the combined effect of baseline SCD-associated leukocytosis and intense systemic inflammation (Rees *et al.,* 2010; Ojor Ileli *et al.,* 2025).

Progression to severe AKI is consistent with the known renal tropism of Lassa virus and immune-mediated tubular injury (Nath & Hebbel, 2015; Guasch *et al.,* 2006; Ashley-Koch *et al.,* 2011). Coexisting sickle nephropathy may have created a particularly vulnerable renal environment. Early initiation of hemodialysis was critical to survival, in keeping with evidence from LF treatment centers in endemic regions (Asogun *et al.,* 2012; Okwuraiwe *et al.,* 2025). Ribavirin, administered with renal dose adjustment guided by adult pharmacokinetic data, achieved viral clearance by

Day 14 despite the lack of pediatric dosing guidance in renal impairment (Eberhardt *et al.,* 2019; Lawal *et al.,* 2024). Persistent electrolyte abnormalities and metabolic derangements likely reflected renal tubular dysfunction, capillary leakage, gastrointestinal losses, and dialysis-related fluctuations (Nath & Hebbel, 2015). Episodes of hypothermia and bradycardia were consistent with severe metabolic exhaustion (Okokhere *et al.,* 2018).
Overall, this case suggests a clinically distinct and exceptionally severe phenotype resulting from the interaction of LF and homozygous SCD. It highlights the need for targeted management guidelines, improved access to renal replacement therapy in endemic settings, and further research into immunogenetic and phenotypic modifiers of disease severity in this high-risk population (Ojor Ileli *et al.,* 2025; Rafiu *et al.,* 2025).

CONCLUSION

Lassa fever in individuals with sickle cell disease is a life-threatening condition with a high risk of severe hemolytic and renal complications. Early diagnosis, prompt initiation of ribavirin, timely renal replacement therapy, and coordinated multidisciplinary care were central to survival in this case. Delayed access to dialysis and specialist care may worsen outcomes in resource-limited settings. Further research is needed to clarify immunogenetic modifiers, pediatric-specific antiviral dosing, and the pathophysiologic interactions between Lassa fever and hemoglobinopathies.

Clinical Implications:

- Children with SCD are high-risk for severe LF.
- Early initiation of renal replacement therapy is lifesaving.
- Multidisciplinary care optimizes outcomes.
- Pediatric antiviral dosing protocols require further research.

Recommendations

1. Early Risk Recognition: Patients with sickle cell disease presenting with suspected Lassa fever should be considered high risk for severe hemolytic and renal complications, warranting early escalation of care.

2. Prompt Renal Replacement Therapy: Timely access to hemodialysis was critical to survival in this case and should be prioritized when acute kidney injury develops in similar high-risk patients.

3. Renal-Adjusted Antiviral Therapy: Ribavirin dosing adjusted for renal impairment, guided by available pharmacokinetic data, was feasible and effective, highlighting the importance of individualized dosing in pediatric patients with renal dysfunction.

4. Early Multidisciplinary Management:
Coordinated care involving infectious disease, nephrology, hematology, and pediatric teams facilitated timely interventions and optimized clinical outcomes.

5. Monitoring for Severe Hemolysis and Metabolic Derangements:
Close surveillance for intravascular hemolysis, electrolyte abnormalities, and metabolic exhaustion is essential, as these complications significantly influenced disease course and management decisions in this case.

Limitations: This study is a single-case report and thus limiting its generalizability. Also, the descriptive nature of the study does not allow for statistical inference. The paediatric-specific pharmacokinetic data for ribavirin in renal impairment are also limited. Additionally, there were limitations of renal biopsy and long-term follow-up.

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REFERENCES

Ashley-Koch, A. E., Okocha, E. C., Garrett, M. E., Soldano, K., de Castro, L. M., Jonassaint, J. C., Orringer, E. P., Eckman, J. R., & Telen, M. J. (2011). MYH9 and APOL1 are both associated with sickle cell disease nephropathy. *British Journal of Haematology*, *155*(3), 386–394. https://doi.org/10.1111/J.1365-2141.2011.08832.X/FULL

Asogun, D. A., Adomeh, D. I., Ehimuan, J., Odia, I., Hass, M., Gabriel, M., Ölschläger, S., Becker-Ziaja, B., Folarin, O., Phelan, E., Ehiane, P. E., Ifeh, V. E., Uyigue, E. A., Oladapo, Y. T., Muoebonam, E. B., Osunde, O., Dongo, A., Okokhere, P. O., Okogbenin, S. A., … Günther, S. (2012). Molecular Diagnostics for Lassa Fever at Irrua Specialist Teaching Hospital, Nigeria: Lessons Learnt from Two Years of Laboratory Operation. *PLOS Neglected Tropical Diseases*, *6*(9), e1839. https://doi.org/10.1371/JOURNAL.PNTD.0001839

Asogun, D. A., Günther, S., Akpede, G. O., Ihekweazu, C., & Zumla, A. (2019). Lassa Fever: Epidemiology, Clinical Features, Diagnosis, Management and Prevention. *Infectious Disease Clinics of North America*, *33*(4), 933–951. https://doi.org/10.1016/j.idc.2019.08.002

Eberhardt, K. A., Mischlinger, J., Jordan, S., Groger, M., Günther, S., & Ramharter, M. (2019). Ribavirin for the treatment of Lassa fever: A systematic review and meta-analysis. *International Journal of Infectious Diseases*, *87*(2), 15–20. https://doi.org/10.1016/j.ijid.2019.07.015

Guasch, A., Navarrete, J., Nass, K., & Zayas, C. F. (2006). Glomerular involvement in adults with sickle cell hemoglobinopathies: Prevalence and clinical correlates of progressive renal failure. *Journal of the American Society of Nephrology : JASN*, *17*(8), 2228–2235. https://doi.org/10.1681/ASN.2002010084

Happi, A. N., Happi, C. T., & Schoepp, R. J. (2019). Lassa Fever Diagnostics: Past, Present, and Future. *Current Opinion in Virology*, *37*(1), 132. https://doi.org/10.1016/J.COVIRO.2019.08.002

Lawal, Q. O., Okoeguale, J., Oiwoh, S. O., Akhigbe, T. G., Eifediyi, R. A., & Okogbenin, S. A. (2024). Addressing bottlenecks in Lassa fever treatment: overcoming the ribavirin parenteral formulation challenge. *Tropical Medicine and Health 2024 52:1*, *52*(1), 46-. https://doi.org/10.1186/S41182-024-00609-0

Nath, K. A., & Hebbel, R. P. (2015). Sickle cell disease: renal manifestations and mechanisms. *Nature Reviews. Nephrology*, *11*(3), 161. https://doi.org/10.1038/NRNEPH.2015.8

Ojor Ileli, S. M., Okosun, O., Dawodu, S. O., Otumu, O. S., Onyerikam, I. H., Ugadu, C. O., Onyeke, I. F., Uanzekin, D., Unaware, E. O., Ehiaghe, E., Apeleokha, M., Ejidike, N. O., Ugbeni, H. E., & Akpede, G. O. (2025). Disease severity and outcome of Lassa virus disease among Nigerian children with Sickle Cell anaemia, an observational study [Conference Abstract]. *Journal of Interventional Epidemiology and Public Health*, *8*(ConfProc5). https://doi.org/10.37432/JIEPH-CONFPRO5-00204

Okokhere, P., Colubri, A., Azubike, C., Iruolagbe, C., Osazuwa, O., Tabrizi, S., Chin, E., Asad, S., Ediale, E., Rafiu, M., Adomeh, D., Odia, I., Atafo, R., Aire, C., Okogbenin, S., Pahlman, M., Becker-Ziaja, B., Asogun, D., Fradet, T., … Sabeti, P. C. (2018). Clinical and laboratory predictors of Lassa fever outcome in a dedicated treatment facility in Nigeria: an observational cohort study. *The Lancet. Infectious Diseases*, *18*(6), 684. https://doi.org/10.1016/S1473-3099(18)30121-X

Okwuraiwe, A. P., Onyeaghala, C. A., Ozoude, O. T., Suleiman, M. O., Abdu-Aguye, S. N., Ezekwelu, N. J., Oyeniyi, T. A., Jegede, A. O., Egwudo, A. E., Okeke, O. P., Abodunrin, O. R., Akinsolu, F. T., & Sobande, O. O. (2025). Clinical outcomes

of Lassa fever in West Africa: A systematic review and meta-analysis. *MedRxiv*, *3*(1), 64. https://doi.org/10.1101/2025.01.10.24319564

Rafiu, M. O., Akerele, N. N., Dada, S. A., Erohubie, C. E., Ahmed, S. D., Edeawe, O. I., Ogbetere, Y. N., Akerele6, J. M., Adetunji, A. E., O. Owhin, S., Osuji, K., Günther, S., Azubike, C. O., Okokhere, P. O., & Akpede, G. O. (2025). Contribution and Characterization of Lassa fever in Severe Acute Kidney Injury (AKI) in Endemic Areas of Southern Nigeria: A Retrospective Observational Study. *SM Journal of Nephrology and Kidney Diseases*, *6*(1), 1–8. https://doi.org/10.36876/SMJNKD.1027

Rees, D. C., Williams, T. N., & Gladwin, M. T. (2010). Sickle-cell disease. *The Lancet*, *376*(9757), 2018–2031. https://doi.org/10.1016/S0140-6736(10)61029-X

Tebbi, C. K. (2022). Sickle Cell Disease, a Review. *Hemato 2022, Vol. 3, Pages 341-366*, *3*(2), 341–366. https://doi.org/10.3390/HEMATO3020024

Ugwu, N. I., Ugwu, G. C., Chika-Igwenyi, N. M., Ugwu, C. N., Akpa, C. O., Ezeokoli, E. O., Nwani, E. I., & Ajayi, N. A. (2023). Successful management of Lassa fever disease in a Nigerian with haemoglobin SC disease at the Lassa fever management centre of Alex Ekwueme Federal University Teaching Hospital Abakaliki, Nigeria. *The Nigerian Health Journal*, *23*(2), 688–692. https://doi.org/10.60787/tnhj.v23i2.662

Uppala, P. K., Karanam, S. K., Kandra, N. V., & Edhi, S. (2025). Lassa fever: A comprehensive review of virology, clinical management, and global health implications. *World Journal of Virology*, *14*(3), 108405. https://doi.org/10.5501/WJV.V14.I3.108405

Yusuf, R., Hassan, A., Ibrahim, I., Babadoko, A., & Ibinaiye, P. (2017). Assessment of kidney function in sickle cell anemia patients in Zaria, Nigeria. *Sahel Medical Journal*, *20*(1), 21. https://doi.org/10.4103/1118-8561.204326