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Journal of Medical Case Reports and Case Series ISSN: 2692-9880

Case Report Volume 6 Issue 02

Case Series On Acute Kidney Injury In Patients With Rift Valley Fever At Mbarara Regional Referral Hospital, Southwestern Uganda

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Received date: 27 March 2025; **Accepted date:** 14 April 2025; **Published date:** 17 April 2025

Citation: Muhindo R, Kansiime G, Kayongo B, Mwine A, Atukwase G, et al. (2025) Case Series On Acute Kidney Injury In Patients With Rift Valley Fever At Mbarara Regional Referral Hospital, Southwestern Uganda. J Med Case Rep Case Series 6(02): https://doi.org/10.38207/JMCRCS/2025/APR06020320

Abstract

Introduction: Rift Valley Fever (RVF) is an endemic zoonotic viral disease which usually affects livestock in parts of Africa and the Middle East. In patients infected with RVF, there is a risk of acute kidney injury (AKI) and acute liver failure (ALF). In this report, we present a case series of four patients diagnosed with RVF infection, highlighting the complications of AKI and ALF, with varying outcomes, including the utilization and impact of dialysis.

Methods: We conducted a retrospective analysis of medical records for four patients admitted and managed at Mbarara Regional Referral Hospital (MRRH) in Southwestern Uganda with confirmed Rift Valley Fever infection. Clinical, laboratory, and outcome data were collected and summarized.

Results: All four patients presented with characteristic symptoms suggestive of RVF, and laboratory investigations confirmed the diagnosis. Each patient had complications of AKI and acute liver failure. All patients required dialysis for renal support; however, logistical issues prevented access to hemodialysis for all but two of the patients. Among the patients who received hemodialysis, both demonstrated recovery from AKI. Unfortunately, the two patients who were unable to access dialysis succumbed to the complications.

Conclusion: RVF infection can lead to severe complications, including AKI and acute liver failure. The timely initiation of dialysis appears crucial for improved outcomes in these cases. However, logistical barriers may hinder access to dialysis, emphasizing the need for improved healthcare infrastructure and accessibility in regions endemic to RVF.

Keywords: Rift Valley Fever, Acute Kidney Injury, Acute Liver Failure

Introduction

Rift Valley Fever is caused by the Rift Valley Fever Virus (RVFV), which is a member of the Phlebovirus genus in the Bunyaviridae family. It was first identified in the 1930s in the rift valley of Kenya as an epidemic on a sheep farm **[3]**. The first outbreak in Uganda was in 1963 and was later followed by the next major outbreak in 2016. In this outbreak, the disease was confirmed from samples of both humans and livestock **[7]** from Kabale in Southwestern Uganda. While RVF primarily affects livestock, the disease is typically transmitted to humans through contact with products of infected animals or through bites of mosquito vectors. It can also be transmitted through the ingestion of meat and uncooked milk of infected animals **[8]**. Veterinarians, abattoir workers and livestock farmers are particularly at risk of infection **[1]**. This risk is particularly significant in Southwestern Uganda where majority of the rural population is involved in livestock farming.

Patients with RVF present with symptoms varying from mild flu-like symptoms to severe cases with about 1% developing hemorrhagic disease. Rift valley fever infection (RVFV) can lead to significant morbidity and mortality in humans and up to 2% of cases may be fatal **[6]**. A systematic review done on the clinical manifestations in RVFV in humans revealed that renal complications mainly presented as acute renal failure **[2, 4]**.

Although the pathophysiology of RVF-associated AKI and ALF is not fully understood, it is believed to be related to infection of the

kidneys and liver by the RVF virus **[5]**. Direct cytopathic effects in the kidneys lead to tubular necrosis and AKI. In the liver, hepatocellular damage and ALF are due to an inflammatory response to infection by the RVF virus.

While RVF-associated AKI is rare, it can significantly impact patient outcomes. The occurrence of both AKI and ALF in patients with RVF is rare but is associated with a poor prognosis, thus the need for early recognition and appropriate management of patients presenting with these complications. Understanding the clinical course and management of these complications is crucial for optimizing patient care.

Methods

The four patients reported in this case series were confirmed as having RVFV infection. These patients were evaluated and managed at the special pathogens' unit of Mbarara Regional Referral Hospital having been received at the emergency department in June and July 2023. This regional special pathogens/isolation unit has the capacity to manage patients admitted with various medical conditions that require isolation. It also serves as the referral centre with specialized care including advanced laboratory services and various chemistry tests. Samples collected from patients suspected to have viral hemorrhagic fevers (VHF) are shipped and tested at the Uganda Virus Research Institute in Entebbe. RVF diagnosis was confirmed using real-time reverse transcriptase- polymerase chain reaction (RT-PCR) testing of blood samples, which detected the presence of Rift Valley fever virus. The turnaround time for results was 48 to 72 hours. Mbarara Regional Referral Hospital also has a dialysis unit that can perform conventional intermittent hemodialysis.

Case presentations

We present a summary of 4 patients who had a PCR-confirmed diagnosis of RVF and were admitted and managed at Mbarara regional referral hospital in southwestern Uganda.

Case 1: NA, a 27-year-old previously healthy male butcher, HIV-negative, who presented with a one-week history of on-and-off high-grade fever associated with malaise, severe muscle cramps, and joint pains. No history of convulsions or altered mentation was reported. Three days prior to admission, he reported Right Upper Quadrant (RUQ) pain associated with jaundice, anorexia, nausea, and non-projectile non-bilious vomiting. No history of hematemesis, diarrhea, constipation, melena, hematochezia or bleeding from any orifices was reported. One day prior to admission, he reportedly passed tea-colored urine, associated with drastic reduction in urine output, but no other urinary symptoms. On examination: An acutely sick-looking young male, in fair nutritional status with moderate scleral jaundice. No other stigmata of chronic liver disease. He was afebrile, with no conjunctival pallor, cyanosis, finger clubbing, edema or palpable lymphadenopathy. No oral lesions or thrush noted. Admission vitals: BP 127/88 mmHg, PR 91bpm, SPO2 96% on room air, respiratory rate 16 breaths per minute & T37.1°C. The Abdomen was soft, with moderate tenderness elicited in the RUQ. He had tender hepatomegaly, two finger-breadths below the right costal margin.

Case 2: AE, a 43-year-old male subsistence farmer with no known chronic illnesses, came in as a referral from a peripheral facility where he had been managed for seven days, he sought care because of a febrile illness characterized with on-and-off high-grade fevers, general body weakness, joint pains and progressive yellowing of eyes of spontaneous onset. During the course of care, the patient also reported oliguria. No history of altered mentation associated with the illness. A day prior to referral, the patient developed epistaxis. No history of hematemesis, diarrhea, constipation or melena. On examination: Adult male, sick-looking, afebrile, not in obvious respiratory distress, with moderate-to-severe scleral jaundice, mild conjunctival pallor, no edema, cyanosis, finger clubbing or lymphadenopathy. Admission vitals: BP 145/95 mmHg, PR 63 b/m, SPO2 97% on room air, Temp 37.2^{o}C.

Case 3: UB, a 55-year-old male, non-diabetic, non-hypertensive, chronic smoker, abattoir worker, presented with two weeks' history of high-grade on-and-off fevers associated with general body weakness, one week's history of blood-stained loose stools 2 to 3 motions per day, associated with abdominal pain, distension and vomiting (contents non-blood stained). Two days prior to admission, he reported reduced urine output and worsening body swelling. On examination: very sick looking, afebrile, arousable, moderate pallor and jaundice, no cyanosis, no finger clubbing, no palpable lymphadenopathy, soft neck, GCS-14/15 with confusion. Per Abdomen: moderately distended with mild generalized tenderness, no organomegaly. The rest of his examination was normal.

Case 4: NK, a 50-year-old male smoker and alcoholic, working as a butcher who presented with a five days' history of worsening epistaxis of spontaneous onset associated with altered mentation and general deterioration of the patient's condition. The illness had started as a febrile illness with on-and-off low-grade fevers, a dry cough and progressive yellowing of eyes which was initially managed as malaria in a lower health facility without improvement. He also reported significant reduction in urine output and passing dark colored urine. On examination: The patient was very sick, afebrile with very dry mucous membranes, had acidotic respiration, moderate jaundice, mild pallor, no peripheral edema or cyanosis. Vitals at admission: SPO2-98% on room air, BP-135/81 mmHg, PR-100b/m, Breathing rate: 32 cycles/min, GCS-12/15. Per Abdomen: Non-tender, no organomegaly. Examination of his chest revealed vesicular breath sounds.

The laboratory investigations and management of the patients is summarized in the table below.

Case	Laboratory Findings	Treatment	Outcome
1.	Creatinine: 8.38 mg/dl (0.6-1.1) Urea:146.9 mg/dl (NR:10- 50) AST: 12235 IU (NR: 0- 37) ALT: 2249 IU (NR: 0- 42) Urine dipstick: Protein ++ Blood +++ Hb: 14.9g/dl (NR: 11.0-16.0) Platelets:59x10^3 (NR: 150- 450x10^3) B/S for malaria: Negative HepBsAg: Negative HCV: Negative RT-PCR: Positive for RVF	IV normal saline 500mls 8 hourly IV Metoclopramide 10mg BD 2/7 IV Omeprazole 40mg OD 5/7 Lactulose syrup 15mls TDS Received 5 sessions of hemodialysis (14th – 18th April 2023) Platelets transfusion - 3 units	Date of admission: 13th April, 2023 Discharged on 24th April, 2023 Repeat laboratory tests on 29th June 2023 Creatinine: 1.28 mg/dl Urea: 25.6 mg/dl AST: 63 IU ALT: 46 IU Platelets: 220,000 Hb: 14.4 g/dl
2	Creatinine:10.2 mg/dl (0.6-1.1) Urea: 182 mg/dl (NR: 15-10) AST: 905.1 IU (NR: 0-38) ALT: 407 IU (40-306) Urine Dipstick: Protein + Blood+++ Hb: 10.3 g/dl (11.5-15) Platelets: 28x10^3 (150-450x10^3) Na^+ 125.9 mmol/L (135-155) K^+ 6.46 mmol/L (3.5-5.5) RT-PCR: Positive for RVF	IV Tranexamic Acid 500mg BD 5/7 IV Vitamin K 10mg stat IV Omeprazole 40mg BD 1/7 PO Omeprazole 40mg BD 1/52 IV Calcium Gluconate 10% 10mls BD 2/7 Tabs Desmopressin 0.2 mg BD Transfusion with 3 units of Fresh Frozen Plasma. Received 4 hemodialysis sessions.	Escaped from the ward, after the 4th session of hemodialysis with significant clinical improvement (resolved epistaxis, urine output 0.7ml/kg/day). Follow-up phone call after 1 week reported the patient resumed his daily activities
3	Creatinine: 13.8 mg/dl (0.6-1.1) Urea: 375 mg/dl (10-50) AST: 1638 IU (0-37) ALT: 947 IU (0-42) Urine dipstick: Protein +++ Blood +++ Hb: 6.0 g/dl (NR: 11.0-16.0) Platelets: 136x10^3 (126-438x10^3) Na: 121.1mmol/l (135-145) K: 7.12 mmol/l (3.5-5.5) RT- PCR: Positive for RVF	IV Actrapid insulin 10 IU in 50mls D50% IV Normal Saline 1 litre stat then, 500 mls 6 hourly for 2/7 IV Lasix 40 mg BD 2/7 IV Calcium Gluconate 10% of 10 ml OD Transfused 3 units of whole blood Was not able to afford dialysis	Died during the course of care, after 5 days following admission
4	Creatinine: 6.24 mg/dl (0.6-1.1) Urea: 194.2 mg/dl (10-50) ALT: 1634 IU (0-42) AST: 5398 IU (0-37) ALP: 630 (80-306) Urine Dipstick: Protein ++ Blood: +++ Neutrophils: 10.71 (1.0-5.3) Hb: 10.4 g/dl (12.2-17.7) Platelets: 55 x10^3 (126-438x10^3) INR (PT time): 1.65 (0.9-1.3) Na 114.3 (135-145) K 4.77 (3.5-5.5) Malaria RDT - Negative RT-PCR: Positive for RVF	IV Ceftriaxone 2g OD IV 3% Hypertonic saline 100mls OD IV Tranexamic Acid 500g TDS IV Vitamin K 10 mg OD IV Normal saline I liter stat, then 500mls TDS for 24hrs Platelet transfusion- 2 units	Date of admission: 16/07/2023 Died 17/07/2023

Discussion

This case series demonstrates the severity of RVF complications involving AKI and ALF. The simultaneous involvement of the liver and kidneys can complicate management and contribute to poorer outcomes. The exact underlying mechanisms of hepatic and renal injury in RVF are not fully understood but they are likely multifactorial and require further investigation. Hypovolemia, direct viral invasion of renal tissue, and immune-mediated damage could contribute to renal injury. According to a 2009 retrospective study at Gezira Hospital for Renal Diseases, Sudan, renal impairment was detected in 60% of patients admitted with RVF, 90% of whom required dialysis. The overall mortality rate was 40%, 31% in patients with acute kidney injury, 25% in those with the hepatorenal syndrome and 31% in patients with primary hepatic involvement and mild renal impairment. **[4]**.

Early recognition of AKI and ALF in RVF patients is crucial for the prompt initiation of appropriate supportive measures. Optimal management involves close monitoring of liver and renal function, adequate fluid resuscitation, and consideration of liver support therapies. Renal replacement therapy may be necessary in cases of severe AKI.

In this case series, all the cases were isolated and provided with supportive care, including hydration, antipyretics, and analgesics. In consultation with the nephrology team, measures were taken to manage AKI, including close monitoring of fluid and correction of electrolyte imbalances. Liver support measures, such as lactulose and vitamin K administration, were instituted for patients with ALF. Patient outcomes varied with two cases (case 1 and 2) showing significant improvement. Both cases presented with stage 3 AKI (oliguric AKI) and required support with acute hemodialysis which they were able to afford. Subsequent follow-up revealed improving kidney function with confirmed laboratory recovery in case 1 and clinical recovery in case 2. Two patients (case 3 and 4) developed progressive worsening of their clinical status and eventually met their demise.

Case 1 and 2 both had risk factors for RVF and presented with non-specific symptoms for RVF. In sub-Saharan Africa, it is not uncommon to treat patients presenting with febrile illnesses for malaria or other infectious diseases like enteric fever. However, in these 2 cases, the index of suspicion for RVF was heightened by the occupational history of the patients and the fact the they presented during a period of an ongoing outbreak.

Case 3 and 4 also presented with RVF associated complications and were unfortunately unable to afford dialysis. Despite conservative management with supportive care, both patients eventually succumbed to the disease. Case 3 had no family member and was managed with the available medications in the hospital. We attempted to correct his hyperkalemia with the available medication but we were unable to repeat or progressively monitor response due to lack of resources. This patient died after 5 days of admission. Case 4 had complicated RVF and had altered mental status on arrival. His initial laboratory assessment also revealed severe hyponatremia which was treated with 3% hypertonic saline. Repeat investigations were unavailable due to limited resources by the family. They were not able to afford acute hemodialysis.

Conclusion

These cases illustrate the importance of considering Rift Valley Fever as a differential diagnosis in patients presenting with fever and a history of exposure to livestock in endemic regions. RVF complicated by AKI and ALF represents a severe manifestation of the disease. Healthcare providers should be vigilant for the development of these complications in patients with RVF. Prompt recognition, appropriate supportive care, and multidisciplinary management are essential for optimizing patient outcomes in such cases. Timely initiation of appropriate therapy including hemodialysis may have a mortality benefit. Access and affordability of such treatment is a significant challenge in resource limited settings. Public health authorities should continue surveillance efforts to detect cases of RVF in the community before the patients get complications like AKI and ALF. In the same vein, efforts to make dialysis more available and affordable to all members of communities remains a key factor in the quest to achieve universal health coverage and kidney health for all.

Statement of Ethics

Written informed consent was sought from 2 of the patients (NK and AE) while consent for 2 of the incapacitated patients (UB and NK) was sought from their care takers. Confidentiality and anonymity of the patients was maintained. Ethical approval was sought and approved by the Mbarara University of Science and Technology Institutional Review Board.

Funding Sources: This case series did not require any funding.

Acknowledgement

We would like to thank the patients and their family members who allowed us to publish this report. We also express our sincere gratitude to the personnel in the isolation unit, the dialysis team and the laboratory staff of Mbarara regional referral hospital for their contributions to offering care to these patients.

Author Contributions

RM is the main author and is the nephrologist and physician in charge of the isolation unit. GK, CM, BK, SA, OK and NW were responsible for managing the patients, AM, GA, SA and MK were responsible for dialyzing the patients. NN supported the laboratory investigations. RM, CM and ES were involved in data collection and writing the summary of the cases.

Conflict of interest: The authors have no conflict of interest to declare

Data Availability: The data is available upon request.

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