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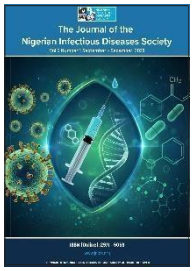

Case Report

A Probable Case of Lassa Fever Occurring Twice in A Patient - A Case Report From The Irrua Specialist Teaching Hospital, Irrua, Nigeria

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ABSTRACT

Lassa fever is endemic in parts of West Africa.

We report the case of a 31-year-old Nigerian who was diagnosed in 2005 with Lassa fever, using the FMOH, Nigeria and The WHO Lassa fever diagnostics guidelines. In 2023, he was again diagnosed with Lassa fever, this time, using RT-PCR. He recovered.

Immune response to Lassa fever is not long lasting enough to protect against a recurrence of the disease.

To the best of our knowledge, there is no previous documentation of a patient who had Lassa fever twice. An attack of Lassa fever does not confer a life- long immunity.

Keywords: Lassa fever, occurring twice, probable case, Irrua.

INTRODUCTION

Lassa fever is a viral haemorrhagic fever transmitted by the 'multimammate rat' (*Mastomys natalensis*),[1]. The modes of transmission include ingestion of contaminated food items, exposure of broken skin or mucous membranes to body fluids, inhalation of virus in aerosols.[2]

About 80% of cases are either asymptomatic or mild, but can be severe in about 15% of Cases[3]. Approximately 15-20% of patients hospitalized for Lassa fever die from the illness[3]. However, in the community only 1% of all Lassa virus infections result in death[3]. Diagnosis of Lassa fever is based on laboratory tests[4]. Treatment of Lassa fever includes supportive care and definitive treatment with ribavirin. Although there are currently no therapeutic trials on the use of ribavirin in Lassa fever, the drug however appears effective, particularly when commenced early in course of the illness, as evidenced by expert opinion and some observational studies, reducing case fatalities[5]. There is currently no licensed vaccine available for Lassa fever prevention.

Lassa fever virus causes suppression of both the innate and adaptive immune responses. It directly infects and disrupts the function of dendritic cells.[6] The infection does not result in the induction of significant amounts of neutralizing antibodies. Such antibodies are only detected in very small amounts during recovery[7]. Antibodies in convalescent serum have been shown to be helpful in survival of patients who are treated with it; however, it is uncertain for how long these antibodies would remain in the serum of survivors, in preventing re-infection or disease. To the best of our knowledge at the time of preparing this manuscript, there is no previously documented case of a patient who had Lassa fever twice. It is on this premise we decided to bring this to the awareness of the scientific world and global academic community. Indeed, in Lassa fever disease,' lightning does strike twice at the same spot'!

CASE SUMMARY (FIRST ADMISSION)

A 31-year-old medical doctor who presented to the accident and emergency unit on 22/06/2005, with high-grade fever and generalized headache of 2 days duration and one episode of non-projectile vomiting, with no heamatemesis. There was no seizure, sore throat or any history suggestive of bleeding diathesis, liver disease or a family history of such. He had parenteral quinine a day prior to presentation, with no relief of symptoms.

On clinical examination, he was acutely ill-looking, febrile, (T=37.8^{0}C), with bilateral periorbital oedema, marked bilateral subconjuctival heamorrhage. His blood pressure was 140/100mmHg. Examination findings of other systems were normal.

At the time of this admission, RT-PCR diagnostic facility was unavailable in the hospital. However, the following investigations were done;

Crude clotting time= 30 minutes (control: < 20 minutes)

Liver function test:
Alkaline phosphatase (ALP) = 63 I.U. (9-35) I.U.
Aspartate aminotransferase (AST) = 43 I.U. (≤ 12 I.U.)
Alanine aminotransferase (ALT) = 34 I.U. (≤ 12 I.U.)
Total protein= 7.4g/dl (6.0-8.0) g/dl
Albumin =4.8g/dl (3.5-5.0) g/dl.
Globulin =2.6g/dl (2.0-3.2) g/dl

Urinalysis: protein +, glucose +, pH=6.0, others=nil.

Random blood glucose: 60mg/dl.
Malaria parasite (microscopy): scanty.

Full blood count:
PCV= 42%, WBC= 7400/mm3, Neutrophils=43%. Lymphocytes = 55%, platelets = 198,000/mm3.

Kidney function test: Na+ = 138mmol/l, K+ = 4.1mmol/l, Cl- = 98mmol/l, HCO3 = 28mmol/l, Urea = 48mg/dl, Creatinine = 1.2mmol/l.

A diagnosis of Suspected Lassa fever was made. He had the following treatment:

I.V Ribavirin 8g loading dose (day 1), then 4g daily from day 2-4, then 2g daily, from day 5-8.

Tab, *Artemether/Lumenfantrine* (80/480) mg, 1 at 0 hr, 8hrs, 24hrs,36hrs, 48hrs and 60hrs.

I.V Ceftriaxone 2g daily for 7 days.

He recovered and was discharged home after 8 days on admission. He had an unremarkable outpatient clinic follow-up visit after 2 weeks.

CASE SUMMARY (SECOND ADMISSION)

A 49yr-old medical doctor who presented with headache of 4 days duration, fever and generalized body weakness of 3 days duration. There was no history of sorethroat, seizures and no history suggestive of bleeding diathesis. He is a known hypertensive, diagnosed 12 years earlier, on amlodipine and lisinopril. Following the onset of symptoms, he took antimalarials (β-Artheeter injection), antibiotics (levofloxacin, ceftriaxone/sulbactam) injections. However, symptoms persisted. A Lassa virus RT-PCR done was positive.

On clinical examination, he was febrile,(Axillary temp=38.2^{0}C), his blood pressure was 175/104mmHg. Clinical examination of other systems was normal.

The following investigation results were obtained:

Full blood count:

PCV=44.7%, WBC= 2100/mm3, neutrophils=51.3%, lymphocytes=38.9%, monocytes=9.8%,
Platelets= 105,000/mm3, Hg=15g/dl.

Random blood glucose: 167mg/dl.

Urinalysis: protein +, glucose +, pH= 6.0, S.G.= 1.030.

Kidney function test:

E/U/Cr: Na+ = 143mmol/l, K+ =4.0mmol/l, Cl- =98mmol/l, HCO3- =14mmol/l, Urea= 25mg/dl, Creatinine= 1.2mg/dl.

Liver Function Test: AST (SGOT) = 141 I.U, ALT (SGPT) = 114 I.U, LDH=1444 I.U, Amylase = 124 I.U, CPK= 697 I.U. Total protein= 7.2g/dl, Albumin= 3.9 g/dl, Total bilirubin= 0. 7mg/dl... CRP= 25.1mg/dl.

He had the following treatment:

I.V. Dexamethasone 4mg 12houly for 72 hrs.

I.V. Ribavirin @ 100mg/kg stat = 7.2g (4.8g stat, then 2.4g after 8hrs) on day 1, then

25mg/kg/day from day 2-6 = 1.8g daily. Then, 12.5mg/kg/day from day 7-10 = 900mg daily.

Tab amlodipine 10mg daily, tab lisinopril 10mg daily.

He made a remarkable recovery. A repeat Lassa virus RT-PCR done was negative. He was discharged home after 17 days on admission.

He had two unremarkable follow-up clinic visits at 2 weeks and 6 weeks post-discharge.

DISCUSSION

In the year 2000, the Federal Ministry of Health of Nigeria (FMOH) developed a diagnostic criterion for Lassa fever[8]. This was done to aid clinical diagnosis as laboratory diagnostic capacities were largely unavailable across the country at the time. It consists of both major and minor criteria for diagnosis. However, in 2010, The World Health Organization (W.H.O) developed a case definition criterion for Lassa fever based on clinical symptoms and laboratory parameters, to assist with the diagnosis[9]. It also has major and minor criteria.

In the first admission, diagnosis of Lassa fever was based on clinical and supportive laboratory and bed side investigations (liver function test, crude clotting time, urinalysis) because there was no facility for RT-PCR diagnostics at the time. Based on the WHO and FMOH, Nigeria, criteria, we strongly think he had Lassa fever. He lived in an urban community endemic for Lassa fever, and worked in a neighboring endemic rural community. He had been in these communities for just over a year, and apparently had not acquired enough protective immunity against the disease. He was then a first- line duty doctor working at the accident and emergency unit of the hospital, where he attended to several febrile patients. He could have contracted the disease in the line of duty or via peri-domestic route. The crude clotting time far exceeded the control, which was expected in Lassa fever. The AST/ALT (SGOT/SGPT) levels were thrice the upper limit of normal, he also had proteinuria. Clinically, he had a high-grade fever which didn't respond to antimalarials (quinine), and also had bilateral subconjuctival heamorrhage. His condition responded to ribavirin, which played a significant role in the management of Lassa fever.

He was diagnosed with Lassa fever using RT-PCR 18yrs later, at the age of 49yrs. It is generally assumed that a single infection of the Lassa virus will produce a life-long immunity.[10] Seropositive individuals in endemic regions have been shown to have Lassa virus-specific CD4+ memory cells, recognizing epitopes in nucleocapsid protein (NP) and glycoprotein precursor (GPC), that are maintained for years after the infection.[11] There is also evidence of secondary infection (seroconversion), with no evidence of progression to disease in individuals who have had a primary Lassa virus infection.[12] Since he first had Lassa fever, he had lived in the same community for 18 years and it stands to reason that he might have acquired some herd immunity following a possible repeated exposures to the virus as a medical doctor.

This immunity from previous infections or disease seems not to be very effective or long-lasting enough to prevent another disease. There is therefore an urgent need to ramp up efforts in vaccine development.

CONCLUSION

Lassa fever remains a disease of public health importance. Improvement in socioeconomic conditions and awareness campaign would positively impact on its prevalence and incidence. Some level of immunity does result from the infection. However, the degree or extent of protection offered is not indefinite. This case negates the age-long assumption that an attack of Lassa fever confers a life-long immunity.

LIMITATION

It is highly regrettable that the blood sample for diagnosis in the first admission was not stored, due to lack of facilities for long-term storage like -80^{0}C freezers in the hospital at the time. And RT-PCR was not performed at the time of infection.

Declarations:

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