

Lassa fever: A case report

***Chundusu C M¹, Isa S E¹, Jonathan B², and Datong P³**

Case Report

ABSTRACT

Objectif: Severe Lassa fever with high mortality among health care providers is usually a human to human infection that requires high index of suspicion to diagnose. This case report is to describe a peculiar case of Lassa fever among health worker.

Result; A severe form of Lassa fever was diagnosed early in a healthcare provider and patient recovered fully.

Conclusion: Fatality can be greatly reduced if early diagnosis as well as prompt specific treatment is instituted.

Keywords: Lassa fever, Jos.

*Corresponding author: Dr. C. M. Chundusu. E-mail: calebchundusu@yahoo.com

¹Department of Medicine, Jos University Teaching Hospital, PMB 2067, Jos, Nigeria

²Department of Family Medicine, Plateau State Specialist Hospital, Jos, Nigeria

³Plateau State Human Virology Research Centre (PLASVIREC), Plateau State Specialist Hospital, Jos, Nigeria

Fièvre de Lassa: Un rapport de cas

*** Chundusu C M¹, Isa S E¹, Jonathan B², Datong, P³**
Rapport de cas

RÉSUMÉ

Contexte de L'Étude: La fièvre de Lassa sévère avec une mortalité élevée chez les fournisseurs de soins de santé est habituellement un être- humain à l'infection humaine qui nécessite l' indice élevé de suspicion pour diagnostiquer.

Objectif: Pour signaler un cas particulier de la fièvre de Lassa Chez travailleurs de la santé.

Résultats: Une forme grave de la fièvre de Lassa a été diagnostiquée tôt et les fournisseurs de soins de santé et le patient se sont rétablis complètement.

Conclusion: La fatalité peut être considérablement réduite si le diagnostic précoce ainsi que le traitement spécifique est exécuté.

Mots Clés: E fièvre de Lassa, Jos.

* Auteur correspondant: Dr. C. M. Chundusu. E-mail: calebchundusu@yahoo.com

INTRODUCTION

Lassa virus is primarily transmitted by a (multimammate) rat specie Mastomys natalensis. The rat is the reservoir host for the virus. Humans get infected when the rodent's excreta contaminate food or other items within the house, the virus can spread secondarily from person to person (1). Most infections are mild and subclinical, severe multi-systemic disease however occurs in 5-10% of infections (2) mostly by human to human. The illness usually begins insidiously with fever, weakness, malaise joint pain or lumber pain cough and severe headache. Pharyngitis often exudative and conjunctivitis may occur early. In severe cases prostration dehydration and facial or neck oedema can occur (3). Laboratory findings include Serum aminotransferases which may be elevated with a pattern similar to that seen in alcoholic hepatitis (AST>>ALT) with lymphopenia, thrombocytopenia and defective platelet function though bleeding is limited in severity. Serum aminotransferases greater than 150iu/l with viraemia usually has mortality rate of about 80%. Deafness may occur in about 30% of patients. About 50% of affected patients will then recover.(4, 5) In Nigeria Lassa fever is said to account for most cases of fever associated with deafness in adults.(4, 5)

CASE REPORT

This was a 36 year old female nurse working at the Accident and Emergency unit who presented to the Accident and Emergency unit of the Plateau State Specialist Hospital in January 2011 with a high grade continuous fever, chills and rigors of 3 days duration. There was severe generalized headache, insomnia and post-prandial non projectile vomiting. She had no neck pain or stiffness. She had earlier on had artesunin combine therapy for three days and had started ciprofloxacin the previous day without improvement.

She was found to be toxic, febrile with a

temperature of 38.0⁰c, weak and dehydrated. She was conscious and alert with no neurological deficit or meningeal sign. She had a respiratory rate of 28 circles per minute and vesicular breath sounds. Pulse rate was 92 beats per minute of regular moderate volume. Blood pressure was 80/60 mm of Hg. The apex beat was at the fifth left intercostals space mid clavicular line and, heart sounds were first and second only. Nothing was found on abdominal examinations.

An initial working diagnosis of typhoid septicaemia was made initially, based on non-responds to antimalarials. She was admitted and started on parenteral artesunate (E-mal®), intravenous ceftriazone and 5% dextrose saline were also administered.

Laboratory results obtained were PCV 33%, WBC = 8.1×10^9 [N 57%, L41%, M 0.1%, E 0.1%]. Blood and urine culture yielded no growth. U/E [Na 120, K 3.8, Cl 96, HCO³ 20, urea 2.7 and Cr 78 all in mmol/l]. Other results are as seen in the tables.

On the fourth day of admission her temperature went up to 40.0⁰c, she became restless, dyspneic. She was flaring and had puffy face. Respiratory rate went up to 33 circles per minute, with posterior basal fine crepitations on auscultation. Pulse rate also went up to 124 beats per minute, blood pressure 100/60 mm of Hg with no added sound on auscultation of the precordium. There was no sign of bleeding bed side whole blood clotting time was greater than 20 minutes.

Diagnosis of Lassa fever was made based on the above findings and treatment was initiated, blood sample was taken to closest virology unit (PLASVIREC). Confirmation was done in the Netherlands. Patient was isolated from other patients and all people including medical staff that got in contact with the patient were advised to report if they had similar symptoms of fever and headache. Parenteral ribavirin was commenced at a dose of 32mg/kg 6hrly start

then 16mm/kg 6hrly for 4days to continue with 8mg/kg for another 6days. She made a dramatic improvement, temperature came down to normal within 72hrs and headache stopped. Ceftriazone and E-mal® were stopped after 72 hours. She got the ten day course of ribavirin and discharged after thirteen days on admission. She had 2 subsequent follow up visits in which she appeared apparently normal, no deafness. The viral study was diagnostic of Lassa fever.

DISCUSSIONS

Lassa fever was first documented in a nursing staff and has remained a major treat to healthcare providers (6). It is again unfortunate that most documented outbreaks of Lassa fever were obtained following the demise of a healthcare provider or a group of healthcare providers, a great human and economic lost.

This disease with very high mortality of about 80% (4) in severe cases is usually associated with high viral load (4,5). A situation commonly seen with human to human transmission in healthcare providers, due possibly to repeated contacts with mild cases. We highlighted some factors that contributed to the success in the treatment outcome of a severe case of Lassa fever.

In this report, the nurse would likely have had repeated exposure to many mild cases of Lassa, a phenomenon seen with many other fatal viral infections like severe acute respiratory syndrome (SARS), bird-flu virus, swine flu virus and more recently middle east respiratory syndrome virus (MERS). Clinical diagnosis was suspected on the third day of admission when features of severe form of the disease started manifesting. Symptoms like persistent high grade fever in which conventional artesinin combination therapy and broad spectrum anti-microbial medications (third generation cephalosporins) could not control and breathlessness suggesting pulmonary oedema though viral myocarditis could have

been a possibility. Prolonged bleeding time and high PTTK (Partial Thromboplastin Time with Kaolin) test (table 1) highly suggested clothing abnormality though there were no obvious bleeding (4, 6). Other factors that made Lassa fever a more likely diagnosis was the season in which the case occurred being dry season (7).

The antibody test to Lassa virus unfortunately does not differentiate active disease process from cured state because an individual who recovers from the illness will still have the antibody circulating in the body for some time (4,5). A local ongoing study in symptomless pregnant women in Jos showed that about 60-70% had antibodies to Lassa virus. National figure documented about 13% prevalence, in the general population. (8)

Despite developing severe features, the patient did survive because ribavirin which is currently the only potent drug against the Lassa virus was readily available. Timely intervention had saved this patient's life. Laboratory result of the PTTK (table 1) showed a return to the normal value following treatment with ribavirin. Similarly slight liver enzymes increment was notice (table 2) which regressed with treatment. Confirmation of the disease was made weeks after discharge.

Health care givers are prone to having severe form of Lassa fever. The success in treatment of Lassa fever depends on early diagnoses which for now rely mainly on high index of suspicion. (9) Mortality can also be greatly reduced when specific treatment with ribavirin is started early for the patient who could be a healthcare provider whenever necessary.

REFERENCES

1. Buckley SM, Casals J. Path biology of Lassa fever. Int Rev Exp Pathol. 1978;18: 97-136.
2. McCormick JB, Webb PA, Krebs. A prospective study of the epidemiology

- and ecology of Lassa fever. J infect dis. 1987; 55-437
3. Holmes GP, McCormick JB, Trock SC. Lassa fever in the United States. Investigation of a case and new guidelines for management. N Engl J Med. Oct 18 1990;323(16):1120-3.
 4. Cummins D, McCormick JB, Bennett D. Acute sensorineural deafness in Lassa fever. JAMA. Oct 24-31 1990;264(16):2093-6.
 5. Jay MT, Glaser C, Fulhorst CF. The arenaviruses. J Am Vet Med Assoc. 2005;227:904-15.
 6. Frame JD, Baldwin JM, Gocke DJ, Troup JM. Lassa fever, a new virus disease from West Africa. Clinical description and pathological findings. Aj trop med hyg. 1970; 19:670
 7. Adewuyi G M, Fowotade A, Adewuye B T. Lassa fever; Another infectious manace. Afr. jornl. of clinical and experimental microbiology 2009; 10(3);244-155
 8. Fed Min of Health Nigeria. Weekly update on epidemics in Nigeria as at 30th march 2012. April 2012.
 9. Isa ES, Shehu NY, Juryul RN, Simji SG. Epidemiological and clinical description of Lassa fever in Jos, Nigeria. High Med Res J 2013; 13:3-7.

Table 1: Clothing profile of the patient during hospital admission.

<i>Clothing profile</i>	DAY 3	DAY 6
PT(prothrombin time)	1' 32"	2' 26"
PTTK(Partial Thromboplastin Time with Kaolin)	52' 6"	1' 50"

PTTK was markedly prolonged and returned to normal with commencement of ribavarin.

Table 2: Liver function tests of the patient during admission

Liver function test	DAY 9	DAY 16
Total protein (g/l)	69	83
Albumin (g/l)	36	33
Bilirubin total (mmol/l)	10.2	10.2
Bilirubin conjugated (mmol/l)	5.1	5.1
Alkaline phosphatase (iu/l)	71	44
Alanine transaminase (iu/l)	19	8
Aspartate transaminase (iu/l)	13	5

No marked liver enzyme derangement seen, the slight elevations regressed.