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## A case report on Lassa fever and hearing loss: A rare occurrence and review of the literature

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### ABSTRACT

Lassa fever is highly endemic in Nigeria and other West African countries, it is a disease associated with high case fatality and chronic sequelae in those that survived. Lack of effective vaccine has made the disease difficult to control and its prevention depends on eradications of the multimammate rats and universal precautions by all when a case is identified. We report a case of a 51 years old health worker who had a severe form of Lassa fever complicated by sensorineural deafness at the University College Hospital, Ibadan, South West Nigeria.

**Keywords:** Lassa fever, viral haemorrhagic septicaemia, sensorineural hearing loss

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## Introduction

Lassa fever is caused by the zoonotic Lassa virus, a member of the arena viridae. It is one of the leading causes of viral haemorrhagic fever in Nigeria and other West African countries. The prevalence of seropositivity of antibodies to this virus was the highest in a serosurvey of five viral agents associated with haemorrhagic febrile infections in different parts of Nigeria.<sup>1</sup>

This disease had been present in Nigeria and other sub-Saharan countries for decades but was first described in 1969 in the town of Lassa in Borno state, Nigeria. Lassa town is located at the Yedseram river valley at the south end of Lake Chad.<sup>2</sup>

There have been frequent epidemics of this disease in Nigeria largely because Lassa virus is borne by multimammate rat (*Mastomys natalensis*) which is ubiquitous in the West African sub-region and quite difficult to eradicate. Humans are infected by contact with the excreta of these rats (virus is constantly being shed in the excreta of these rats) or by eating the rats. Transmission from person to person has also been established, presenting a challenge for health care workers and people who are in the same household with the affected person. The disease affects all age groups. Severe multisystemic disease occurs in about 20% of individuals affected while 80% have mild or no observable symptoms.<sup>3</sup>

Sensorineural hearing loss among other neurological sequelae, has been found to occur in 25% of confirmed cases of Lassa fever.<sup>4</sup> The hearing loss occurs typically during the convalescent stage of the disease, however hearing loss in the acute phase of the infection has also been documented.

In this environment, reports of hearing loss in confirmed cases of Lassa fever are few and none has been reported in our institution hence, we present a case of sensorineural hearing loss in a medical practitioner with confirmed diagnosis of Lassa fever.

## Case report

A 51-year-old male medical practitioner presented to the medical emergency room with

high grade intermittent fever, passage of dark coloured urine, myalgia, anorexia and malaise of two weeks duration. He had been treated for malaria fever and sepsis at a secondary health care facility without improvement. He had treated patients with fever, jaundice and conjunctivitis prior to the onset of his illness.

Examination revealed an ill looking patient, conscious and alert, dehydrated, febrile with a temperature of 39.6°C and bilateral conjunctivitis. There was no evidence of pharyngeal inflammation and no petechial haemorrhages seen. Examination of his chest and abdomen revealed essentially normal findings.

Investigations carried out included a full blood count which revealed a packed cell volume of 41%, white blood cell count of 27,500/mm<sup>3</sup> and platelet count of 395,000/mm<sup>3</sup>. Retroviral screening was non-reactive for HIV I and II, a quantitative analysis of Glucose 6phosphate dehydrogenase showed reduced activity while the clotting profile was normal. Urinalysis revealed protein 4+ and blood 3+ in the urine, an abdominal ultrasound scan done showed grade I parenchymal disease.

A clinical diagnosis of severe sepsis with a yet to be identified focus complicated by intravascular haemolysis was made and he was placed on intravenous ceftriaxone. A close differential diagnosis of acute glomerulonephritis was entertained.

While on admission he developed anaemia (packed cell volume 24%), increase in leucocytosis (white blood cell count 33,600/mm<sup>3</sup> as well as thrombocytosis (platelet count 508,000/mm<sup>3</sup>) and he was transfused with two units of whole blood.

Blood culture remained sterile after 7 days of incubation. Blood sample was taken and sent to the laboratory for Lassa virus because of our high index of suspicion due to the endemicity of haemorrhagic fever in this environment particularly Lassa fever and the fact that there was a preceding history of exposure to patients with fever and jaundice and he was also not responding to antibiotic therapy. He developed bilateral hearing loss on the 6<sup>th</sup> day on admission. This progressively worsened till it became profound and could only depend on lip reading

for conversation 48 hours after. There were no other otologic symptoms. Pure Tone Audiometry revealed severe to profound sensorineural hearing loss bilaterally. Figure 1.

The Lassa virus was identified by Reverse transcriptase polymerase chain reaction (RT-PCR) and the patient was commenced on Rivabirin on the 10<sup>th</sup> day on admission. He thereafter began to have progressive clinical improvement and was discharged home after 25 days on admission. However the hearing loss persisted.

A trial of intra-tympanic Dexamethasone (8mg) was given twice but there was no noticeable improvement in the pure tone average. Patient is, at present, being considered for cochlear implantation.

## Discussion

This case has shown the transmission of Lassa virus infection to medical personnel in a person to person mode of transmission. The circumstance might suggest that it was acquired in the community; this could be from the patients seen during consultation or otherwise. This case buttresses the fact that Lassa fever is highly contagious and that health care givers are at increased risk of being infected particularly in areas of high endemicity. The effort to prove this will depend on a careful molecular epidemiological surveillance in the community and contact tracing with viral studies. Another peculiarity about this case is the fact that deafness presents early in the disease, acute phase. This is unusual in that most literature reports deafness in the convalescent period.<sup>3</sup> The occurrence of deafness in the acute period as seen in this case suggests increased severity of the disease. Lassa fever affects between 100,000 and 300,000 people annually in West Africa of which about 5000 die.<sup>3</sup> The neurological sequelae of this infection include encephalitis, meningitis, seizures and unilateral or bilateral deafness.<sup>3</sup>

Lassa fever causes deafness more than other viral haemorrhagic fevers such as Yellow fever, Ebola and Marburg. Deafness as a complication of Lassa fever was first reported in 1972.<sup>5,6</sup> Other otological manifestations that have been reported include tinnitus, autophony, vertigo,

nystagmus and ataxia.<sup>7,8</sup>

Deafness is the most common neurological complication of Lassa fever.<sup>3</sup> Varying degrees of deafness occur in approximately one third of cases and in many cases hearing loss is permanent. Severity of the disease does not affect this complication: deafness may develop in mild as well as severe cases.<sup>9</sup>

Moreover, this complication tends to occur in the convalescent phase of the disease although there have been cases where deafness occurs at the acute phase of the disease as was seen in this index case.<sup>10</sup> Viral infections that cause inner ear disorders such as deafness do so in the acute phase of the disease. During this phase, these infections lead to loss of hair and supporting cells of the cochlear as well as disruption of the tectorial membranes and atrophy of the stria vascularis leading to end-vessel thrombosis and inner-ear fibrosis. The vestibulocochlear nerve integrity may also be compromised as a result of direct invasion of the spiral ganglion by the viruses.<sup>11</sup>

Often they result in sudden sensorineural hearing loss (SNHL) and, because of limited diagnosis, most are classified as idiopathic. About 57% to 60% of patients are known to recover spontaneously during convalescence while the remaining ones develop permanent hearing loss.<sup>12</sup>

This typical mechanism of inner ear injury ear does not seem to be the pathogenesis of deafness in patients with Lassa fever as majority of the hearing loss occurs in the convalescent phase of the infection. Moreover, further deterioration in hearing has also been demonstrated even after full recovery from the infection, hence acute immunological response to viraemia may not be responsible for the hearing loss in this infection. It has been postulated that immune response against inner ear structures through molecular mimicry may be the underlying factor for the hearing loss.<sup>9, 13</sup>

Rivabirin therapy when commenced early has not been shown to offer protection against development of sensory neural deafness.<sup>9</sup> This further lends credence to the possibility that the hearing loss is immune response mediated.

Research has also shown that the strain of Lassa viruses found at the West African coast varies in its amino-acid sequence (genome) and tends to elaborate exaggerated immune responses, involving high titres of IgG and IgM<sup>14, 15</sup>. This increases the risk of inhabitants of endemic areas of Lassa fever with sub-clinical infections from previous exposures to stand a higher risk of developing SNHL during their lifetime.

Other mechanisms, which require further research, have also been suggested as the cause of deafness in Lassa fever rather than immune response as cases of unilateral hearing loss from this infection have been reported as against the bilateral condition which would be expected if it were solely from immune response.<sup>13</sup> The treatment of sensorineural hearing loss from Lassa fever has not yielded encouraging results. These treatments have essentially tried to increase the oxygen perfusion and reduce inflammation to the ear. Systemic as well as intratympanic steroids have also been tried with no clear evidence of efficacy. Whereas these treatments have been shown to improve hearing thresholds in patients with idiopathic sudden sensorineural hearing loss.<sup>17,18</sup>

Intra tympanic steroid therapy has been shown to improve the hearing threshold in patients with sudden idiopathic sensorineural hearing loss. This procedure was undertaken after the patient failed to respond to systemic steroid therapy.<sup>18</sup> It can be an alternative for patients in whom systemic steroid therapy is contraindicated or an addition to systemic steroid therapy when indicated.<sup>19</sup> This treatment however did not appear to be of benefit to the patient as there was no improvement in his hearing threshold.

## Conclusion

We conclude that this case has further reinforced exposure risk of healthcare personnel to Lassa fever in the endemic areas and that it could be complicated by hearing loss irrespective of the severity. The hearing loss can occur either in the acute phase or convalescence phase of the disease. The poor response to available treatments makes it pertinent to emphasise universal precautions by all health care personnel when attending to all patients.

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