A CASE OF LASSA FEVER IN PAKISTANI SOLDIER ON UNITED NATION MISSION IN LIBERIA

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INTRODUCTION

Lassa fever is an acute viral haemorrhagic fever endemic in West Africa. It has been reported from Sierra Leone, Guinea, Liberia, and Nigeria[1].. Lassa fever is also the most common hemorrhagic fever that is exported beyond its endemic area to Europe and other parts of the world[2]. One Such case of a Pakistani soldier on united nations mission in Liberia is reported.

CASE REPORT

A 26 year old Pakistani soldier who had recently come to Liberia from Pakistan to perform his duties in United Nations' Mission in Liberia (UNMIL).

He was posted at Bopolo camp in Boomy County when he developed fever with rigors and chills, headache and one episode of vomiting on 4 April 2006. There was no complaint of sore throat, chest pain, abdominal pain or diarrhoea. He was transferred to Pak Level-2 Hospital at Tubmanburg on the same day. At the time of admission to this hospital, his body temperature was 100^0F and BP 110/70 mmHg. Rest of his clinical examination was unremarkable. His investigations including Haemoglobin (Hb), Total Leucocyte Count (TLC), Chest X-Ray (CXR), Liver Function Tests (LFTs) including Aspartate aminotransferase (AST) and Prothrombin time (PT) on the first day are shown in table.

As a number of cases of Lassa fever had occurred in Bopolo camp about two months earlier and some of them had concomitant Falciparum malaria, strict laboratory and isolation protocol for lassa fever was adopted from the time of admission of this patient. He was started Quinine for Falciparum malaria and broad spectrum antibiotics empirically for concomitant bacterial infection in addition to symptomatic treatment. His blood parasite density, Complete blood counts (CBC), LFTs, AST, Alanine aminotransferase (ALT), Lactic dehydrogenase (LDH) and PT were monitored regularly. He started having temperature spikes up to 102 F after admission. On the 3rd day his Red blood cells (RBCs) were cleared of falciparum rings but his condition started deteriorating. He appeared very sick and started having vomiting. His temperature remained in the range of 101 F- 102 F, his BP was 90/60 mm of Hg but rest of his systemic examination remained unremarkable. His TLC and Platelets started decreasing and AST started increasing.

On the 6th day of disease his condition deteriorated further. His investigations on this day are shown in table. He was suspected to have Lassa fever and after taking his blood sample for PCR test of Lassa virus, he was started Ribavirin infusion 1800 mg stat, then 900mg 6 hourly for 4 days, then 500mg 8 hourly for next 6 days. His condition deteriorated further on the next day. He started vomiting persistently, his temperature spike went to 103F. Then he started showing dramatic improvement after 48 hours of starting Ribavirin. On the 3rd day of Ribavirin infusion his temperature started settling, his vomiting started decreasing and he complained of epigastric pain. His BP was 110/60 mmHg and he had mild epigastric tenderness. Rest of his clinical examination was unremarkable. His investigations on this day are shown in table.

On the 4th day of Ribavirin therapy (9th day of disease), his fever settled, his vomiting stopped and he felt much better. However, his Hb dropped to 11.5g/dl and platelets showed a rise i.e, 72×10^9/L. Ribavirin was stopped on the 16th day of the disease when its' 10 day course was completed. His laboratory investigations were repeated which are shown in table. During the last seven days of Ribavirin

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treatment he remained asymptomatic. His PCR report received from Department of Virology, Bernhard-Nocht-Institute for Tropical Medicine, Hamburg, Germany confirmed his Lassa fever.

At the completion of Ribavirin treatment his serum and urine samples were sent to Germany for status of viremia and viriuria which turned out to be positive. Then he was shifted to an isolated convalescent room where he remained for about six weeks. Two days after completion of Ribavirin therapy, he developed generalized pruiritus which responded to a course of oral antihistamines. Two days later he developed generalized lymphadenopathy which regressed after three weeks. During these six weeks he kept on having occasional complaints of dyspepsia, insomnia and myalgias which responded to symptomatic treatment. His laboratory tests remained normal during this period. After two months of start of his disease he was discharged from the hospital with advice to abstain from sexual activity or use condom for one more month. Before discharge, his clinical examination and laboratory investigations revealed no abnormality and there was no evidence of sensorineural deafness.

His contacts were traced and were kept under surveillance for three weeks but none of them developed temperature.

DISCUSSION

Lassa fever is seen in all age groups. In 80% the disease is sub-clinical but the remaining 20% have a severe multi system disorder. Typically, after an incubation period of about 10 days (range 3 to 21 days), there is insidious onset of fever, headache, generalized weakness, and malaise. The initial presentation is often non-specific, and the differential diagnosis includes common febrile illnesses found in West Africa including malaria, typhoid, and bacillary dysentery. These conditions have to be ruled out or treated empirically.

Within a few days, these symptoms may be followed by sore throat, cough, retrosternal chest pain, conjunctivitis and abdominal pain. The patients with florid Lassa fever may then manifest facial and neck swelling, sub-conjunctival hemorrhage and bleeding. Pleural effusions are common. Patients with severe illness may deteriorate rapidly, progressing to shock, respiratory distress, coma, seizures, and death. About 15% to 20% of hospitalized patients may die. The overall mortality of Lassa fever is about 1%. Surviving patients generally begin to defervesce within 10 days of onset. With the exception of sensorineural deafness, recovery is usually complete.

Abnormalities in laboratory tests include thrombocytopenia, lymphopenia, proteinuria and elevated blood urea nitrogen (BUN). The AST is often elevated, and higher values are

Table: Results of Investigations

	1st day of admission	6th day of admission	8th day of admission (3rd day of Ribavirin)	16th day of admission (10th day of Ribavirin)
Haemoglobin	13.7 g/dl	14.1 g/dl	13.8 g/dl	14.5 g/dl
White cell count	3.1 x 109 /L	1.8x109/ L	3.1x109 L	5.5x109 L
Platelets count	157x10 9/L	72x 10 9/L	60x10 9/L	386x10 9/L
Blood Slide	Falciparum rings seen	No Falciparum rings seen	-	-
Alanine Aminotransferase	Normal	66 U/L	120 U/L	68 U/L
Aspartate Aminotransferase	Normal	133 U/L	303 U/L	55 U/L
Lactic Dehydrogeanase	-	1180 U/L	1278 U/L	-
Prothrombin time	Normal	Normal	20/14 Sec	Normal

predictive of a poor prognosis[3]. There is disproportionate elevation in AST compared to ALT. Viremia occurs throughout the acute febrile period. In survivors, this viremia is cleared by about 2 weeks. Acute Lassa fever can be diagnosed by ELISA for Lassa IgM antibody and antigen. IgG antibodies are present in almost all patients by 3 weeks and last for years. The virus can also be detected by reverse transcription polymerase chain reaction (RT-PCR). Virus has also been isolated from serous effusions and other body tissues[4].

All persons suspected of Lassa fever should be admitted to an isolation facility and their body fluids and excreta properly disposed of. Supportive treatment consists of adequate hydration, symptomatic measures and proper management of blood losses, shock and superimposed infections. There is decrease in cardiac index and increase in peripheral vascular resistance and poor response to fluid infusion. So, fluids should be used with caution in supporting the circulation. Transfusions are needed occasionally. Secondary bacterial infections usually respond to antibiotics.

Lassa fever can be treated effectively with ribavirin. Ribavirin decreases mortality when started early in the course of disease. If given within six days of the start of illness it may reduce deaths by 90%. Patients with poor prognostic indicators (e.g, AST >150 units/mL) should be treated with intravenous ribavirin given in a dose of 30 mg/ kg initially, 15 mg/kg every 6 hours for 4 days, and 7.5 mg/kg every 8 hours for 6 more days. Modest anemia may occur from hemolysis and reversible normoblastic maturation arrest. In selected patients with overwhelming infection, exchange transfusion of whole blood may be tried[5].

To prevent person to person transmission of Lassa fever during acute illness, barrier nursing precautions should be enforced and needle precautions should be emphasized. Gloves, masks, white coats, and goggles are advised while caring for a sick person. Sterilization of all materials leaving a patient's room including adding disinfectant to toilets before use is recommended. Clinically well patients leaving the hospital are not generally contagious. Patients may transmit virus to spouses in convalesence so condoms should be used during sex for 3 months. Lassa virus has been isolated from urine for several weeks, and the use of disinfectant in the toilet bowl before voiding is advised.

Lassa fever is a preventable disease. Currently, there is no vaccine for Lassa fever, and no experimental vaccine has completely protected nonhuman primates against a lethal challenge[6]. So measures should be taken to keep rodents out of homes and to protect food from the rats in endemic areas. Medical authorities responsible for health care of foreign missions in endemic areas should educate their troops on preventive measures. All contacts of Lassa fever cases should monitor their own temperature daily for three weeks and report regularly to medical authorities. Ribavirin prophylaxis has been offered to contacts at highest risk who had been directly exposed to a patient's body fluids and blood.

As Falciparum malaria and Lassa fever coexist in these endemic areas, one should be watchful of both.

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