Case Report

Rabies: A Novel Clinical Presentation

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Abstract

Rabies is a fatal disease. Saliva of a rabid dog is a rich source of rabies virus. We report a patient who suffered of rabies, who was infected by abrasion caused by the nails of a rabid dog. Dogs often lick their nails and thereby transfer the rabies virus-contaminated saliva to their claws. Despite treatment in our Intensive Care Unit and application of various pharmacological antidotes, we were unable to prevent the fatal outcome.

Keywords: Elapid venom, GABA, glycoprotein, NMDA, rabies

Introduction

Rabies a fatal neurological disease caused by lassavirus. Virus is transmitted through the infected saliva of canine animals. Prevention of this grave disease is achived by pre and post exposure immunization, and rabies immunoglobulin against rabies when it required. Irrespective of freely and free available rabies vaccine at government hospital including primary health centers . Still we witness the grave disease with fatal outcome, because of century old myth deeply rooted amongst villagers that dog bite only village healers (*Tantrik*, *Mantrika* and *ozas*) or herbal remedies can cure and protect from rabies. Rabies virus outer cover is glycoprotein responsible to induce slow apoptosis of infected brain neuron. Rabies encephalitis is easy to diagnose. Even at advanced tertiary care center the fatality of rabies victim is 100%, only in a immunized victim there are chances of recovery with neurologoical deficit.[1] Irrespective of raised titer of rabies antibodies in a infected victim with disease did not arrest the severity of disease. In rabies prevention is a mother of cure.

Case Report

On May 3, 2011, at 10 AM, a 50-year-old female was admitted to an outpatient department hospital at Mahad in the district of Raigad. She complained of insomnia, suffocation, fever, severe whole-body pain, and malaise over the past 4 days. On April 30, she had been examined by her family doctor. She received an unidentified injection, acetaminophen tablets, and oral chloroquine. There was no improvement of symptoms.

Since she additionally complained of chest pain, she was admitted to the Intensive Care Unit (ICU) on May 2, where her pain was attributed to unstable angina. She was closely monitored and was given nasal oxygen, intravenous fluids, a nitroglycerin drip and furosemide. In addition, a treatment with low-molecular-weight heparin 60 mg every 12 h digoxin, aspirin, clopidogrel and statin was started. There was no clinical improvement. On the 3rd day, the ICU doctor transferred her to a tertiary care hospital for further cardiac investigation to rule out ischemic cardiomyopathy.

Instead of going to Mumbai, the relatives brought the patient to the clinic at Mahad. On admission, she was fully conscious. She presented with a history of mitral valve replacement in 1994. She complained of excessive thirst and being unable to drink water for 2 days. While examining her, we asked for a glass of water. Simple by hearing the word water (*Pani* in vernacular language), she suddenly developed severe laryngeal spasm [Figure 1, Video 1]. This repeated laryngeal spasm in response to the word "water" confirmed that she was suffering from rabies. We asked her and the relatives regarding any history of a dog bite. She reported that 4 months ago, a furious dog had bitten four persons from her village. All of

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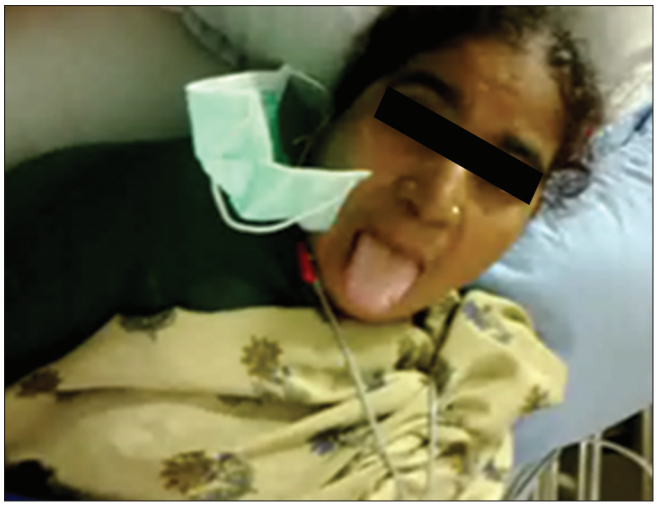

Figure 1: The patient developed laryngopharyngeal spasm just by hearing the word "water" (*Pani* in vernacular language)

them were treated with immunization as exposure prevention by the government hospital. All the four are healthy at present. The furious dog had caught her sari (cloth), and with its claws, it caused multiple abrasions at her right foot. She cleaned the blood and the abrasions by rubbing it with her sari. She and her relatives believed that only the bite of a dog can cause the disease and hence avoided postexposure rabies immunization though it is free and freely available at the primary health center, which is 5 km away from her village. However, she reported that instead of visiting the hospital, she visited a Tantrik (village healer) who gave her some herbal medicine.

Her blood pressure was 140/80, she had no signs of myocardial failure, a grade 3/6 systolic murmur was heard at over the aortic root area, and an ejection click of the mitral valve was heard over the apex. The respiratory rate was 14/min and no pathological breathing sounds were heard over her chest. Her body temperature was 99°F. There was no any neurological deficit except laryngeal spasm.

As major symptom, she described that she experienced tingling and numbness and heaviness in her right lower limb. Troponin T was negative, and serum sodium was 128 mEq/l. Electrocardiogram showed a heart rate of 75 beats/min with minor ST-T changes [Figure 2]. Hemoglobin level was 14.6 g/dl, leukocyte count was 11,100 cu.mm, and blood sugar was 105 mg/dl.

She was isolated in a dark room. Very carefully, we placed a nasogastric feeding tube. She was given intravenous fluids, intravenous atropine 3 mg, magnesium sulfate (MgSO4) 2 g, midazolam 1 mg/kg body weight, and sustained acting zolpidem 12.5 mg through the nasal tube. The next day she developed a mixed form of delirium with being fully conscious in-between (lucid interval). She still occasionally developed laryngeal spasms. On the 3rd day, she presented with tachycardia, delirium, internuclear ophthalmoplegia, and plantar flexion, and the strength in all limbs was grade 5/5,

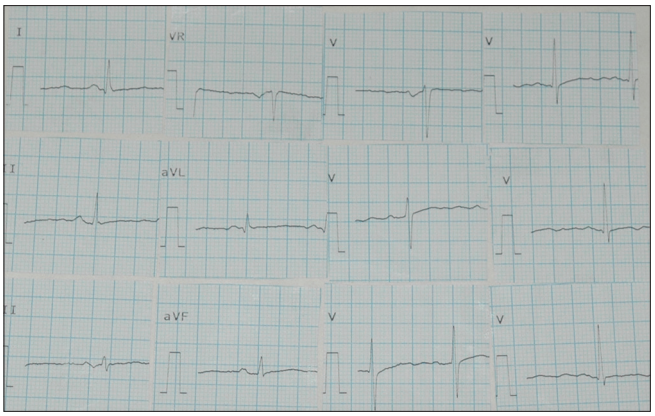

Figure 2: Electrocardiogram – normal sinus rhythm minor ST-T changes

but she had no neck rigidity. Moist rales were heard over the chest, and she became tachypnoeic. At 3 AM on the 4th day, she developed pulmonary edema and massive hematemesis and died of respiratory arrest. She was conscious till death. No attempt to resuscitate her was initiated following the decision of seniors and relatives.

Discussion

Yearly 30,000 victims die of rabies in India alone. The high incidence of rabies in a developing country is due to the mere neglect of rabies immunization after dog bites. This is explained by false beliefs and lack of social awareness though the rabies immunization is given free at primary health centers. Moreover, medical professionals maintain false silence react restrainedly toward this century-old grave disease as seen in the present case.[1] Rabies is a 100% fatal disease; up till now, only 7 cases recovered with prolonged intensive care, irrespective of treatment with antiserum, antiviral, interferon-alpha, corticosteroids, and other immunosuppressant drugs.[1] Dogs often lick their nails and claws where the virus-contaminated saliva remains and can cause severe infections with rabies by abrasions as observed in the present case.[1]

Cellular receptor dysfunctions caused by the virus play an important role in the pathogenesis of rabies encephalitis.[1] Datura seeds (atropine) counteract the rabies virus action as mentioned in *Ayurveda*.[2] In the present case, atropine prevents the development of recurrent severe spasm due to the attempt to swallow the accumulated saliva in the mouth cavity.

The rabies virus binds to the postsynaptic acetylcholine receptors and competitively blocks cholinergic ligands. The chemical and structural sequence homology of the glycoprotein of rabies viruses is similar to the elapid venom neurotoxin alpha-bungarotoxin; it is not available. Molecular scientists should search out the chemical structure from alpha_bungarotoxin, which may competitively inhibit the action of virus glycoprotein or immunoglobulin against alpha-bungarotoxin may neutralize the action of glycoprotein on

infected brain neuron and upgrade the neuronal apoptosis, which is downgraded by virus glycoprotein. The virulence of virus inversely related to the neuronal apoptosis.[1,3,4]

N-methyl-D-aspartate (NMDA) subtype R1 and gamma-aminobutyric acid (GABA) have been suggested as possible rabies virus receptors in the central nervous system. MgSO4, a selective noncompetitive antagonist of NMDA receptors, may prevent cellular injury. MgSO4 prevented sudden cardiac arrest due to cardiac arrhythmias, convulsions, and hypertension in the present case. Zolpidem induces selective inhibition of the normal GABAergic inhibitory neuron. Pathophysiology evoked by rabies virus is its action on NMDA and GABA receptors. Hence, we used these Zolpidem and also MgSO4 as an antagonist to the NMDA receptors.[5]

The chemical sequence homology of the glycoprotein of the rabies virus mimics the krait venom, a neurotoxin called alpha-bungarotoxin, a presynaptic acetylcholine receptor destructor. It is possible that a drug derived from krait venom may act as a competitive antagonist of the rabies virus glycoprotein evokes potent lethal action in central nervous system and peripheral nicotinic receptors. It so might help to inhibit the progress of rabies. However, monovalent krait antivenom is a rich source of alpha-bungarotoxin antibodies, may help to neutralize the rabies virus glycoprotein.[6,7]

Immediate wound cleaning by soap and water and active immunization prevent this grave disease. Chances of development of mild diseases and recovery are higher in partially immunized victim.[1]

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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