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CASE REPORT- FATEL CASE OF RABIES

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ABSTRACT

Rabies is an acute viral encephalomyelitis that is almost invariably Fatal. It is caused by rhabdovirus of the genus Lyssavirus and is endemic in wild and domestic animals in many parts of the world. Areas with high risk of human infection are south- and south-east Asia (Nepal, India), but regions with increased risk also exist in Africa, South America and Eastern Europe. In this article we describe the clinical presentation of a Fatal case of rabies in a young adult of 24 yrs old.

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INTRODUCTION

On 20 April 2016, a 24 year-old male was referred to our hospital Shri Mehant Indresh Hospital from Saharanpur District of UP. He had been bitten in his left leg by a stray dog in Saharanpur four weeks previously. He sought medical assistance in Saharanpur immediately after the incident. Rabies vaccination was not initiated at that time. His symptoms started on 15 April (day 1) with paraesthesia and severe pain in the left leg. The patient presented at a local hospital in Saharanpur on 16 April with fever, nausea, paraesthesia of the left leg, headache and difficulties in swallowing. On the basis of the anamnesis, a rabies infection was suspected, and vaccination was immediately started according to the World Health Organization (WHO) rabies postexposure prophylaxis (PEP) protocol (Rupprecht *et al*., 2002) with purified chicken embryo cell vaccine (PCECV, Rabipur, Behring, "Essen-scheme") and anti-rabies hyper-immunoglobuline (HRIG, Berirab, Behring; 20mg/kg).

Treatment and clinical course

Five days after the onset of illness, the patient was transferred to our hospital Shri Mehant Indresh Hospital. A heminested RT-PCR for rabies virus nucleocapsid RNA from two different saliva samples and one corneal swab that was initiated on the same day, was positive. Antibodies against rabies virus could not be detected in the serum or in the cerebrospinal fluid (CSF), neither by indirect immunofluorescence test (IIFT) nor by ELISA. Patient developed continous seizures in our hospital which were not relieved by anti epileptics.Subsequently, deep analgosedation with ketamine, midazolam and respirator therapy was initiated on day six. Except for amantadine, no other antiviral drugs were given. The patient was enterally nourished, and low molecular heparin in a prophylactic dosage was initiated. Electroencephalography (EEG), did not show significant pathology. Until eight days after onset of symptoms, no rabies virus antibodies were detectable in serum and cerebrospinal fluid. In order to support seroconversion of the patient, a live attenuated rabies virus vaccine (VirBac, Nice) was administered intradermally. On day 11, increasing rabies virus antibodies were detectable by IIFT in the serum, but not in the CSF. A revaccination with the same vaccine was given on day 15 in the hope to induce an earlier antibody response. On day 20, we were able to detect an early antibody response in the CSF also. On day 21 the patient developed pathologically raised blood pressure, his pupils became wide, non-reacting and oval.

No electrical brain activity could be detected by EEG from day 22 onwards. Whether the suppression of electrical brain activity was due to the high dose of sedative drugs or to brain damage caused by the virus is uncertain, and therefore could not be interpreted as a sign of brain death (De Tourtchaninoff *et al*., 1999). The patient was continuously on ketamine for its postulated antiviral potency, even after EEG monitoring showed no electrical brain activity. This therapy was not discontinued because transient EEG findings conclusive with brain death had been described before in the single survivor of a rabies infection (Willoughby, 2007; Willoughby, 2005; Rupprecht *et al*., 2002; CDC, 2004). An MRI scan on day 29 showed a generalised oedema of the whole cortex and the basal ganglia. Although ultrasound examination displayed a normal heart function, the patient developed ventricular arrhytmia before death. He died on day 31 after first onset of symptoms from multi-organ failure.

DISCUSSION

To date there has been only one report of a recovery of a patient with a human rabies infection (CDC, 2004). The therapeutic strategy that had initially been chosen in this case, had failed in other patients. We therefore felt that we had to modify our therapy. We analysed the transplantation-associated rabies cases in Germany in 2005, a treatment trial of a patient in Thailand 2006, and the case of the rabies survivor in Wisconsin, and developed a vaccine strategy that was aimed at inducing an early antibody response. We administered the vaccine intradermally, hoping to achieve better antigen presentation and response in the skin. In addition, this gave us the opportunity to monitor immune response and viral load in skin biopsies. The concept of deep analgosedation with ketamine was chosen because of a lack treatment alternatives and drugs with the potential to suppress viral spread in tissue cultures, even though this had failed in other treatment trials. Ribavirin was not given due to its lack of anti-rabies activity and its potential to delay cellular immunity (Warrell *et al*., 1989).

Despite the fact that we were able to detect an early antibody response in the serum, and later in the CSF, the viral spread in the brain tissue could not be stopped. Since the experiences made in the case of the single survivor were not able to improve the outcome of other cases treated similarly, human rabies infections must still be viewed as always Fatal. We tried to induce anti-rabies antibody production earlier in the course of illness with the idea to have an immune response against virus infected nerve cells before all neurons are infected. In our patient, this strategy failed.

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