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Accepted 01 November, 2012

Rabies is a viral infection of the central nervous system, contracted from the bite of an infected animal, and is nearly always fatal without proper post exposure prophylaxis. Our patient, a 12 year old girl who was bitten by a stray wild dog two month before the onset of a progressive encephalopathy, which began with fever temperature (38.3°C), vomiting, irrational talks, diaphoresis, restlessness, gradual inability to talk, obtunded and altered sensorium. Dirty wound was observed on the site of dog bite; cerebrospinal fluid chemistry showed raised protein (113mg/dl) and glucose (6.6mmol/l). Rabies virologic and serologic testing was not performed due to lack of facilities. She was suctioned regularly, placed on antibiotic, nasogastric tube feeding, anti tetanus serum and anti rabies vaccine. Human rabies immune globulin, (HRIG) was not available for use and she was subsequently discharged against medical advice.

Keywords: Human clinical rabies, Management dilemma, Paediatric department, Adamawa state, North-Eastern Nigeria.

INTRODUCTION

Rabies, an incurable disease feared since antiquity is caused by rabies virus, which is classified in the family Rhabdoviridae and belong to the genus Lyssavirus (Brooks et al 2001). Rabies virus is a neurotropic ribonucleic acid virus transmitted to humans through the saliva of infected animals, usually from bites; the virus is almost invariably fatal after the onset of neurologic symptoms (Jackson 2000). Jackson in (2002) reported an estimated 50,000 cases of rabies in humans occur worldwide each year, with the majority occurring in developing countries and originating from infected dogs. Children aged 5–15 years are at particular risk (Brooks et al 2001). Rabies an enzootic disease of both wild and domestic animals is believed to be the tenth most common cause of death in humans, despite this, rabies is grossly underreported in many countries (Brooks et al 2001).

McDermid et al (2008) in Canada reported 27 cases of rabies in humans over an 80-year surveillance period; they also found that canine rabies was well controlled,
but that due to bat exposure was increasing. The incubation period in humans is typically 1–2 months but may be as short as 1 week or as long as many years up to 19 years; it is usually shorter in children than in adults (Brooks et al 2001). The clinical spectrum can be divided into three phases: a short prodromal phase, an acute neurologic phase, and coma. The Centers for Disease Control and Prevention (2010) published that only six human patients worldwide recovered from rabies and most of them had moderate to profound neurologic sequelae. Because rabies has long incubation period, patients usually present late to hospitals. Most of them present in acute neurologic phase or coma as is the case with our patient. The diagnosis of rabies in index case was clinical, based on exposure history, and compatible clinical manifestations similar to what was published by other investigators (Brooks et al 2001, and McDermid et al 2008).

CASE REPORT

Our patient is a 12 year-old girl who was bitten by a wild stray dog about two months prior to her illness. She first had fever, and vomiting, and talks irrationally. Few days later her symptoms intensified, patient started foaming with excessive saliva from the mouth, became restless, unable to talk, obtunded and her consciousness was clouded. No dyspnoea, convulsions, diarrhea, change in urinary frequency, colour or volume. Traditional medications were administered to her at home before presenting to the hospital without significant improvement. She resides with her parents in a rural community of Adamawa state; her mother is a milk seller while the father is a herdsman. On examination, she was febrile 38.3°C, diaphoresis, and altered sensorium with Glasgow coma scale of 10. A dirty wound was found on the posterior aspect of her left leg. Cerobrospinal fluid chemistry showed elevated protein (113mg/dl) and glucose (6.6mmol/l). Nuchal skin biopsy, saliva and serum samples for rabies virologic and serologic testing were desired; however lack of facilities hindered us from doing these tests.

Patient was suctioned regularly, placed on antibiotic, nasogastric tube feeding, anti tetanus serum and anti rabies vaccine. Wound debridement and dressing were commenced. She did not benefit from Rabies Immune Globulin, Human (HRIG) because of its unavailability. On the fourth day of admission, she was discharged based on request from her parent despite medical advice.

DISCUSSION

The clinical manifestations observed in our patient were similar to other reports on rabies across the world (Jackson 2000, and Jackson 2002). For suspected rabies exposures, bite, wound or exposed surface should be immediately irrigated and washed with soap and water and wound closure should be avoided. Antibiotics and tetanus prophylaxis should be given as necessary. Post exposure prophylaxis should be given to people who have not been previously vaccinated against rabies (Brooks et al 2001, and McDermid et al 2008). Our patient was from a remote village in Adamawa state whose parents may not have enough knowledge on rabies and its prevention. Since the incubation period of rabies could take long period of time, rabies illness may not be considered to be from past exposure. Alternative treatment is often sought elsewhere before presenting to a hospital. Traditional care was given to our patient without improvement before she was brought to the hospital. All these put together could have been responsible for the primary delay in presenting to the hospital that was noted in our patient. However, she was commenced on antibiotics and tetanus prophylaxis in the hospital.

Cases of rabies involving healthy domesticated animals, such as dogs, cats or ferrets that can be observed for symptoms for 10 days, post exposure prophylaxis may be deferred until signs of rabies develop in the quarantined animal or until the testing on the euthanized animal’s brain is negative (McDermid et al 2008). If the animal remains healthy for 10 days, post exposure prophylaxis is usually not required. Our patient was bitten by a wild stray dog that disappeared subsequently, in such scenario; post exposure prophylaxis would have been the best option because the vaccination status of the dog could not be ascertained. Similar view was shared by many authors elsewhere, and they added that people who are bitten in the head or neck by a domesticated animal should also start post exposure prophylaxis immediately; and stop treatment if rabies in the animal is ruled out (McDermid et al 2008).

Clinical rabies is characterized by progressive encephalitis and death, and therapy is largely palliative. Although survival has been reported in some rabies cases, it is pertinent to note that all that survived had received either pre or post exposure prophylaxis (Madhusudana et al 2002). Progressive encephalitis could have been ongoing in our index case because she deteriorated in clinical status that culminated to a state of altered sensorium and was unable to speak. Furthermore, the outcome may not be encouraging due to the fact that she had never received either pre or post exposure prophylaxis for rabies. Reasons for the high case-fatality rate with rabies are not clear (Jackson et al 2003). Some workers, however, attributed the deaths due to rabies to result from respiratory paralysis, which is frequently associated with bats (McDermid et al 2008, and center for disease control 2010). Our patient did not present with respiratory distress probably because dog bite which is not known to cause respiratory paralysis was responsible for her illness. Weli et al (2006) linked early phase of rabies with severe neuronal dysfunction,
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and little neuronal cytopathic damage. Since the pathophysiology of rabies virus infection appears to be primarily neuronal dysfunction rather than inflammation and cell death, the clinical syndrome of rabies encephalitis is theoretically reversible. A fundamental requirement for recovery would be viral clearance and development of a protective immune response. On the basis of this, interferons, ribavirin, and other drugs were used but have shown no beneficial effects (Brooks et al 2001). Ribavirin was not prescribed to our patient because of the aforementioned reason.

Willoughby and colleagues (2005) described a protocol for treating rabies encephalitis that included therapeutic coma, antiviral therapy and intensive care support. The strategy, called the Milwaukee Protocol, involves administration of ketamine, midazolam, amantadine, ribavirin and phenobarbital (Willoughby et al 2005). The premise is that, given sufficient time, antiviral and antiexcitatory therapy will allow for viral clearance and permit clinical recovery. Using this protocol entails the use of paediatric intensive care and prolong ventilator support which are lacking in our facility. This is one reason our patient did not benefit from this protocol.

Another is that some of the drug requirements for the Milwaukee protocol are difficult to get in our health center, possibly because they aren’t the routine essential drugs commonly used. Thirdly, it is sad to note that the use of Milwaukee Protocol did not yield significant results in most rabies cases.

Public health officials are notified of clinical cases of rabies; however, this system of surveillance seems to be weak in our own health system. Possibly the public health department is poised to tackling other diseases like meningitis, polio and cholera that are more common than rabies, and could lead to greater patients disability, morbidity and mortality. Nonetheless, however, contacts of our patient were evaluated for possible post exposure rabies prophylaxis. Fortunately enough contacts of our index case did not meet the criteria for post exposure rabies prophylaxis.

CONCLUSION

Rabies remains an important public health issue. There is need for continued vigilance and public awareness, education of health care workers, and prevention with early post exposure prophylaxis when indicated, all of which are proven to prevent clinical rabies.

REFERENCES


