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Human Rabies: An Exceptionnal Fatal Case Report with Unusauall Manifestations Associated to Encephalitis

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Abstract **Case Report**

Human rabies is considered by the World Health Organization as a public health problem in several developing countries of the world, including Morocco. Diagnostic tools do not make it possible to detect the virus before the disease phase and the prognosis remains fatal once established. The clinical signs of rapidly progressive acute encephalitis in the context of post-exposure to a bite from a sick dog or other animal help guide the diagnosis. Furthermore, the diagnosis can be difficult if the bite is not mentioned or looked for or dates back several months to the illness and also if the clinical presentation is not suggestive. We report a unusuall clinical case who had an atypical presentation with multiple organ symptoms associated to encephalitis.

Keywords: Human rabies, zonoosis, encephalitis, myocarditis, orchitis, post exposition prophylaxis.

INTRODUCTION

Rabies is a zoonotic disease caused by an RNA virus of the Lyssavirus genus; it is transmitted to humans accidentally by the virulent saliva of infected domestic or wild animals. It is responsible for a rapidly progressive acute encephalopathy often progressing to death [1, 2]. Although it has become exceptional in developed countries as a traveler's disease form, human rabies remains a real public health problem in developing countries where approximately 59,000 deaths per year in Africa and Asia are recorded [3]. In Morocco, the number of deaths from human rabies was 15 cases in 2017 and 20 on average per year [4].

It can be highly prevented by post-exposure prophylaxis (PEP) [5]. It is a notifiable disease and considered a biological weapon [2].

We report an unusual fatal clinical case with an atypical clinical symptoms associating encephalopathy, myocarditis and preceded by acute orchitis making diagnosis more difficult to confirmed.

CASE REPORT

A seventeen-year-old male adolescent with no medical history was admitted to the emergency room for acute dyspnea that had been present for less than twenty-four hours. A day before he came for sudden onset of testicular pain explored by testicular ultrasound which was without abnormality and treated with paracetamol and bed rest. He is the third of three siblings. No known pathology in the family. He is never operated on. His psychomotor development was normal. He is not known to smoke or consume alcohol or illicit drugs. Clinical examination in the emergency room found a patient conscious but agitated, polypneic at 32 cycles per minute, his temperature was 37.5°C, his oxygen saturation had increased from 88% to 98% after high concentration mask oxygen therapy, Auscultation found a few crackling rales on both pulmonary fields with absence of cardiac murmur or added noise. He had no turgor of the jugular veins or lower limb edema. Questioning with his family did not reveal any use of toxins or medication other than paracetamol for the treatment of scrotal pain.

The biological assessment found white blood cells at 8000/mm3 including 6500 neutrophils and 1500 lymphocytes. Active protein C was 19. Troponin was 850 pg/ml. Chest radiography (Fig 1) revealed an interstitial syndrome in the perihilar region without any image of nodules or pleural effusion. The electrocardiogram (Fig 2) revealed sinus tachycardia at 140 c/min. Transthoracic ultrasound showed global

hypokinesia of the left ventricle probably related to acute myocarditis with an ejection fraction around 30%. The latter gradually improved after few days. Liver assessment, renal function and hemostasis assessment were normal. A search for respiratory viruses was carried out by a multiplex PCR (herpes, VZV, influenza, RSV, etc.) And a Covid PCR was negative.

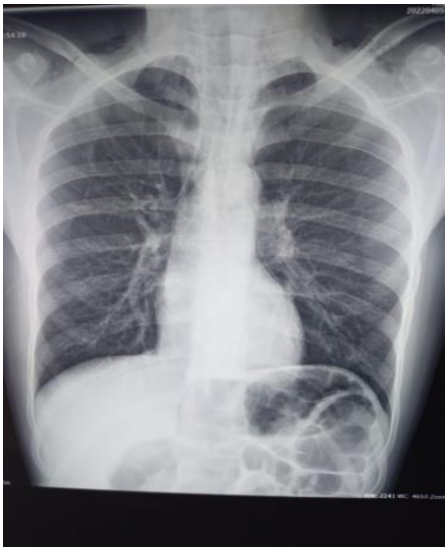

Figure 1: Pulmonary X-ray showed an interstitiel syndrome in the perihilar region without nodules or pleural effusion

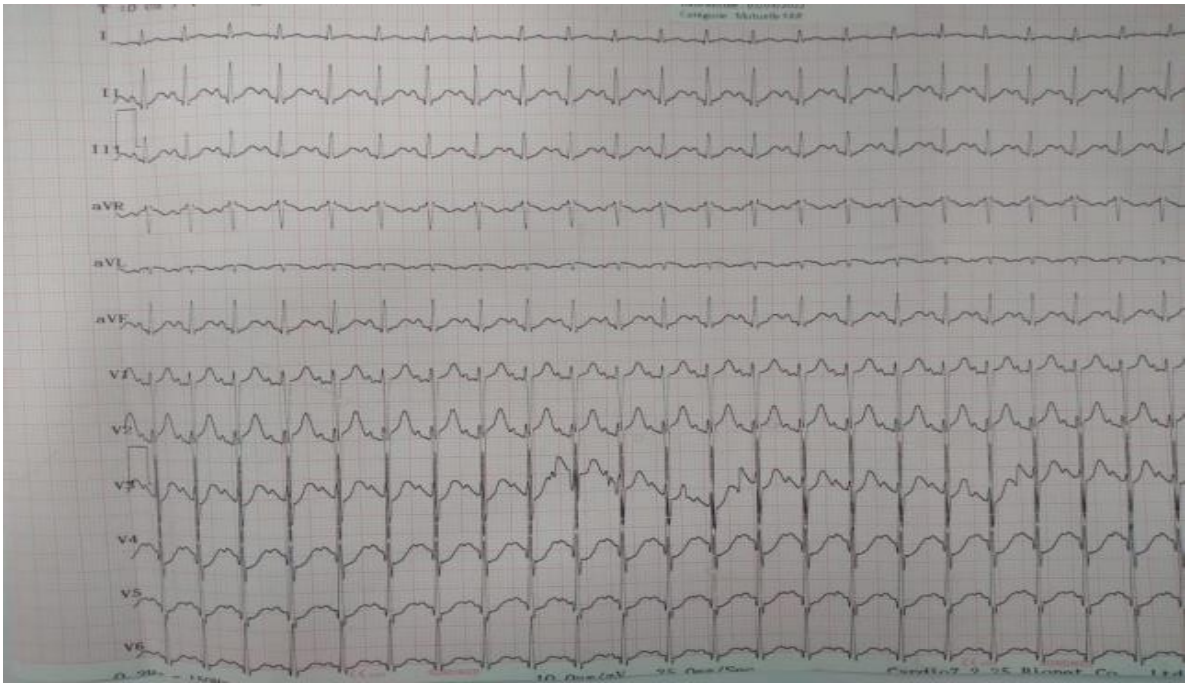

Figure 2: Electrocardiogram showed sinus tachycardia

The patient was admitted to intensive care and put on oxygen therapy and diuretics with improvement on the respiratory level. Furthermore and after a few hours of admission, the neurological state gradually deteriorated, he became more agitated and aggressive requiring the use of bolus of benzodiazepines (midazolam 5mg) several times, there were any epileptic crisis. The examination did not find any sensory or motor deficit. Muscular strength and osteotendinous reflexes were preserved. A brain CT performed under sedation showed no abnormality (Fig 3). The brain MRI did not show signs of encephalitis (Fig 4). Electroencephalogram was normal. The patient was intubated and placed under deep sedation for 48 hours. The biochemical and bacteriological analysis of the cerebrospinal fluid was normal (Proteinorrachia at 0.41 g/l, glucorrhachia at 0.8 g/l). Viral serologies: hepatitis A, B and C, TPHA-VDRL, mumps, EBV and CMV were negative. The autoimmunity test was negative. Blood, urine and gastric toxicological samples were negative.

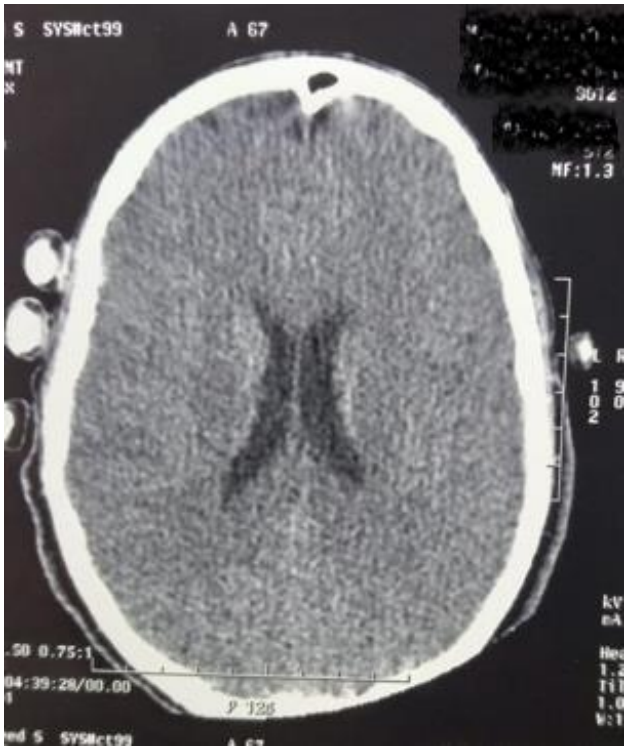

Figure 3: Cerebral computed tomography showed no abnormalities

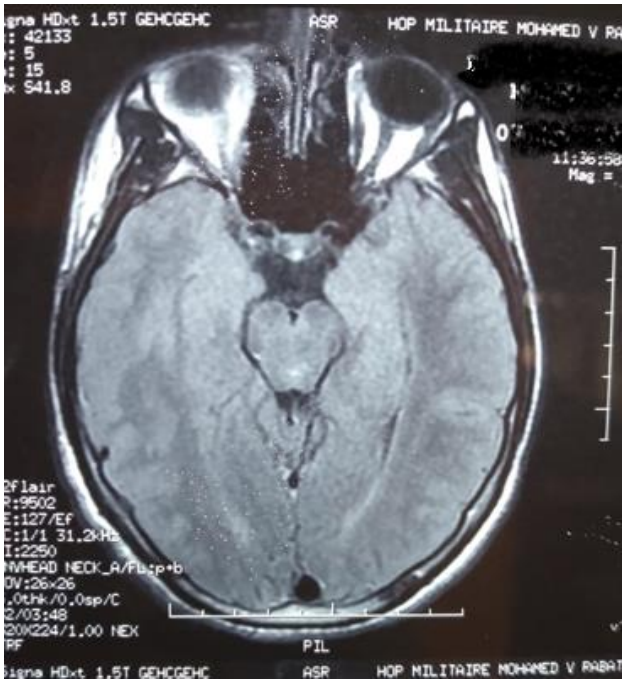

Figure 4: Mmagnetic resonance imaging showed abnormalities

After 3 days, the sedation was interrupted, the patient kept in coma. clinical examination showed laryngeal spasms and hypersialorrhea without hydrophobia led us to ask about the notion of an history of dog bite before, which the family confirmed. This accident had occurred two months before. The patient was bitten on the right foot causing a scar (Fig 5) for which he received local care with anti-rabies vaccination and serotherapy according to his family. Confirmation of human rabies was achieved through positive testing for virus in cerebrospinal fluid. Saliva and skin biopsy from the cervical region were negative. The patient died after eight days in intensive care following an onset of neuro-vegetative disorders. The case was reported to the competent ministerial authorities in accordance with the mandatory reporting laws for this disease.

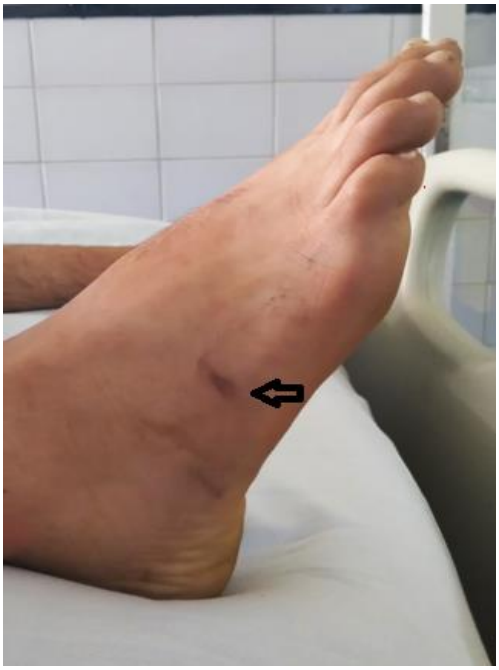

Figure 5: Scar after a stray dog bite 2 mounths ago

DISCUSSION

Human rabies is considered a traveler's disease in Western Europe. In Canada, one case over 15 years has been recorded and 35 cases in the USA over 42 years [5, 6]. In sub-Saharan Africa, the annual number of cases of human rabies declared by the WHO is 0.01 to 3 cases per 100,000 inhabitants. [7]. In Morocco, the number of cases recorded is 20 cases on average per year of human rabies (transmited 94% by dog, 5% by cat, 1% by other animals) and 250 cases on average of animal rabies (134 cases in 2019) [4]. Humans are transmitted by exposure to saliva through bites or scratches (from dogs, bats, cats, jackals, etc.) or infected brain tissue. The incubation period varies from 21 days to 96 days depending on the initial viral load and the number and location of scratch and bite lesions [1, 2, 6, 9-12].

The diagnosis is made at two levels. The first is that of exposure in order to quickly assess the need for post-exposure prophylaxis, and the second is that of rabies disease itself [13]. Clinically, human rabies is present in 2 distinct major forms, and around 65% will develop the furious type and the remaining paralytic rabies. Other forms of presentation include multi-organ involvement, renal failure, acute respiratory distress syndrome, pericarditis, and myocarditis [2]. Our patient had multiple organ manifestations: cardiac, respiratory, testicular and neurological.

Also, agitation, confusion, agressivity and hydrophobia wich is pathognomonic and aerophobia were all reported. Phobic spasms in response to tactile, auditory, visual, or olfactory stimuli pose high moralities within days without intensive ventilatory support. The pathophysiology for the characteristic aggressive behavior in the encephalitic form of rabies remains unknown [14].

Sometimes the diagnosis is made postmortem (38% cases in the USA) [2]. The diagnosis is suspected in the face of a positive exposure investigation, the onset of a rapidly progressive acute encephalopathy. It is confirmed by the search for the virus in saliva, cerebrospinal fluid, a skin biopsy using an RT-PCR (reverse transcription polymerase chain reaction) technique and its antibodies in the blood [2]. There are no means of diagnosis before the disease phase.

The diagnostic difficulty in our case was due to the onset of viral myocarditis before neurological disorder. Viral screening for other viruses was negative, hence the probability that the myocarditis was due to the rabies virus. The notion of testicular pain before clinical worsening increases the probability of a transient acute orchitis caused by the rabies virus but couldnt be confirmed by testicular biopsy.

Therapeutically and once the disease has become symptomatic after a variable period of time, it is essential to emphasize that there is no antiviral or other therapy to kill or neutralize the rabies virus in the brain [15].

The WHO has established a systematic post-exposure prophylaxis (PEP) protocol. It consists of cleaning the wounds immediately and rigorously with water and soap for 15 minutes, receiving an anti-rabies vaccination (double injection on day 0 in the deltoids, on Day 7 and on Day 21 and depending on the indication, administering anti-rabies immunoglobulins or monoclonal antibodies [3]. This protocol is effective in preventing the development of rabies disease, however

some rare cases of failure in the USA concern major bites at the neck, face which are areas of nerve density and short axonal length with a short incubation period [2].

In African series, Post exposure prophylaxis does not provide guaranteed protection from the disease and several authors report failures with death of patients receiving previous prphylaxis of up to 100% [1, 9, 10, 14]. In our patient, despite a PEP protocol, the evolution was fatal.

CONCLUSION

Human rabies remains a public health problem in developing countries. In Morocco, a rabies control program has been established for years with the hope of eradicating the disease. Given its fatality, post-exposure prophylaxis makes it possible to change the course of the disease. Primary prevention against zoonosis involves vaccination of domestic animals and the eradication of sick wild animals.

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