Case Report

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Neuroleptospirosis Masquerading as Guillain Barre Syndrome: A Case Report

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ABSTRACT

Leptospirosis is an anthropozoonosis due to spirochetes. The contamination is direct or by contact with rodent's infected urines.We report an original case of a 61-year-old man, who presented for 3 weeks, flaccid para-paresis, with feverish icterus and red-porto urine. Electromyography objectified acute motor demyelinating polyradiculoneuropathy. Albumino-cytologic dissociation was found in the cerebrospinal fluid. MAT and ELISA's serology returned positive to Leptospira Australis. Anti-GM1 Antibodies were positive. Patient was treated by cephalosporin and immunoglobulin with clinical improvement. Leptospirosis with Guillain-Barre syndrome like with anti-GM1 antibodies are rarely reported. In front of any clinical suspicion, the PCR and the serology by micro-agglutination constitute successive means for diagnosis.

Keywords: Guillain-Barre Syndrome Like; Neuroleptospirosis; Flaccid Paraparesis; Acute Polyradiculoneuropathy; Martin et Petit Microagglutination

Abbreviations: ENMG: Electroneuromyography; CSF: Cerebro-Spinal Fluid

Introduction

Leptospirosis is recognized as the most widespread zoonosis worldwide, affecting man after a contact with affected rat's urine, and also a major cause of disease in many domestic animal species. It was isolated about 100 years ago, as the causative agent of Weil's disease. In the second immune phase of the disease, aseptic meningitis occurs in 80%, with benign clinical course. More severe neurological complications in leptospirosis are rare.

Case Report

A 61-years-old man, with medical history of arterial High blood pressure, type 2 diabetes, dyslipidemia and Myocardial infarction, admitted for weakness of both lower limbs with fever. Signs started 3 weeks before his admission, during a stay in village, by the appearance of fever with yellowish sputum for which the patient was treated by amoxicillin and clavulanic acid, without clinical improvement. Three days later, the patient presented rapidly progressive weakness of both lower limbs with diffuse myalgia, making him bedridden. There was no urinary dysfunction, or diarrhea, or vomiting, or history of vaccination, or trauma.The general examination found conscious patient, jaundiced, febrile to 38.5 °C with red-porto urine. He was well oriented in space nd time, with supple neck. Neurologic examination found flaccid paraparesis quoted to 2/5 with abolished tendon reflex. There were no sensitivity disorders and no paresis in upper limbs. Abdominal, respiratory and cardiovascular examinations were normal. During the first week after admission, the patient presented an ascent of the paresis to both upper limbs with hypo tony, respiratory and gulps disorder. He was transferred to intensive care for two days.The clinical syndrome was acute symmetric ascending flaccid

paraparesis with areflexia, so an electroneuromyography (ENMG) was performed. The ENMG objectified prolonged distal motor latencies, with reduced motor amplitude and temporal dispersion of compound muscle action potential; conduction block was established in the study of right medianus nerve conduction, with increased F-wave latency in all studied nerves.

The sensitive amplitude was normal. We conclude to demyelinating motor polyneuropathy (see table). Cerebro-spinal fluid (CSF) study revealed albumino-cytologic dissociation (CSF-protein=0,9g/l; cellularity=0/mm3). The clinical presentation, neurophysiologic finding and CSF study were compatible with Guillain-Barre syndrome. Nevertheless, There was biological inflammatory syndrome, neutrophil leukocytosis with deep regenerative anemia (Hb=6g / dl), liver cytolysis (100 times the upper limit of normal) and cholestasis, acute renal failure, elevated creatine phosphokinase (95 times the upper limit of normal) and LDH due to rhabdomyolysis. Infectious etiology was suspected, thus multiple serologies were performed and went all negative (hepatitis B and C, HIV, syphilis, herpes simplex virus 1 and 2, varicella-zoster virus, human herpes virus type 6, Epstein-Barr virus, cytomegalovirus, Mycoplasma pneumoniae, Chlamydia pneumonia). Phthisiologic and toxicologic tests went negative (tuberculosis eliminated). In front of the feverish jaundiced state with negativity of exhaustive tests, leptospirosis was evoked. The leptospirosis serology by ELISA and Martin Petit microagglutination technique were positive to "leptospira australis". Also, ELISA test was performed to antiganglioside antibodies, and was positive for anti-GM1 antibodies. The patient was treated by intravenous human immunoglobulin, 3rd cephalosporin generation, Furosemide, hydration schema, transfusion of two blood units and rehabilitation program.

They had led to apyrexia, regression of myalgia and gulp disorders, weakness recovery in upper limbs, as well as the normalization of the biological parameters: renal and liver function, and rate of LDH and CPK. The patient had paraparesis quoted to 4/5 as sequela. The final diagnosis was: acute demyelinating polyradiculoneuropathy secondary to leptospirosis with anti-GM1 antibodies.

Discussion

Leptospirosis, one of the first anthropozoonosis, is an emergent disease. Its annual incidence over the world is about 100.000 cases and about 1000 deaths, essentially in tropical zones [1]. The reservoir is constituted by wild mammals, especially rodents, and domestic mammals as cattle, pig and feline. Some professional activities (sewer workers, breeders) or leisure activities (rafting), expose the skin or mucous membranes to contaminated water.Leptospirosis causes vasculitis explaining the polymorphic and aspecific signs but most of the patients remain asymptomatic [2]. After seven to ten days of incubation, clinical signs appear as fever and visceral involvement: Their most frequent localizations are hepatic revealed by an icterus, renal (50-80 % of the cases) and meningeal (15-20 %). The hemorrhagic syndrome favored by thrombopenia associated with leukocytosis, appear in 70 to 80% of the cases [3]. Neurological disorders reported in patient having leptospirosis include stroke, polyneuropathy, transverse myelitis, Guillain-Barré syndrome, mononeuritis and acute aseptic meningitis [4]. Both direct effect of leptospira and immune-mediated injuries of central and peripheral nervous system are presumed pathogenic mechanisms, almost they have not been completely elucidated [2]. Leptospires occur diffuse vasculitis responsible for the most neurological syndromes in the initial phase, while circulating immune complexes could be associated the others syndromes observed in the second (immune) phase [5].

Antiganglioside antibodies are producted secondary to molecular mimicry between infectious agent and ganglioside. Conduction block are the result of activating complement in contact with antibodies binding to nodes of Ranvier. Such pathogenic mechanisms explain some of neurological syndromes in leptospirosis including Guillain-Barre syndrome like. Nevertheless, few of these cases are reported with objective presence of anti-ganglioside antibodies [5]. The immune mediated process was strongly shown in our case, with compatible clinic and neurophysiologic findings with positive of anti-ganglioside antibody. The second argument of this hypothesis is the favorable evolution after immunoglobulin therapy.Our case illustrates a Guillain-Barre like syndrome. Rare cases are reported elsewhere. Sudden onset paraparesis with leptospirosis was reported in a 26 year-old woman. She presented renal failure with hyperkalemia after a febrile episode with diarrhea. A case of infection with Leptospira icterohaemorrhagiae followed by renal failure and ascending polyneuropathy was reported in a 65 year-old woman [6]. Same case with presumed infection with Leptospira serovar Copenhageni was reported in a 12 year-old girl [7]. Jaundice with renal failure and flaccid paraplegia was cited in a pediatric case [8]. In most of these cases, the diagnosis was based on the IgM serology, without identifying the infection serovar [9]. Leptospira is a negative gram spiral bacteria, mobile, extracellular, belonging to the order of Spirochaetales.

Several diagnostic methods are available. The highlighting of leptospira in plasma, urine or CSF, in the direct examination in the dark field microscopy, is exceptional [10]. This is due to mediocre sensibility (10.000 leptospira by milliliter being necessary to establish the diagnosis) [11]. The culture is possible but difficult and needs long period. The useful sampling must be made during the first ten days of evolution for blood and CSF; and during the second to third week for urine. In practice, culture reserved for specialized laboratories. The PCR allows a fast and reliable diagnosis especially in early phase, but it is available only in rare centers. In numerous clinical circumstances, serology stays the key of diagnosis. Indeed, most of the time in these paucisymptomatic pictures where leptospirosis is late evoked, the previous techniques are negative, insensitive or unavailable. Sev-

eral serologic techniques are available but the reference technique remains the microscopic agglutination test (MAT), requiring the maintenance of about twenty strains and reserved for specialized laboratories. Various dilutions of patient's serum are incubated with several live leptospirosis serovars. Agglutination is visualized in dark field microscopy. Indeed, except the endemic zones, a title upper or equal to 200 in the presence of suggestive signs defines a probable case. In endemic zones, the required title must be upper or equal to 800 [12]. In practice, the early serum is negative half the time and thus imposes a late serum control two to three weeks later.

In summary, PCR and serology must be considered as successive means of diagnosis, the serology having the advantage of its availability and its sensibility since it is repeated. In spite of the absence of consensus, the first line treatment of leptospirosis is Amoxicilin (intravenous) during seven days. Hypersensitivity reaction secondary to massive bacteria lysis (Jarisch-Herxheimer reaction) can arise at patient treated by penicillin [13]. Cephalosporin third generation gives variable effectiveness but present the advantage of broad spectrum of action and unique daily administration for the Ceftriaxone [14]. Penicillin G as well as cyclin and macrolides, are alternatives in moderate to severe forms [15].

Conclusion

Our case illustrates the clinical polymorphism of leptospirosis. Clinicians must be aware of the possibility of its unusual features, because early diagnosis and adequate treatment improve the prognosis. Leptospirosis must be considered for people exposed to rodents, suggesting the interest to include its serology. On the other side, In front of ascent flaccid paraparesis with areflexia, extra-neurological symptoms and biologic findings are helpful for differential diagnosis of Guillain-Barre syndrome. This emphasizes the relevance of targeting tests to objectify the causal agent in front of atypical presentation.

Conflict of Interest

The authors declare having no conflict of interest.

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