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Case Report

Guillain Barré Syndrome and Hepatitis A: A Case Report

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Abstract

Introduction: Guillain Barré syndrome (GBS) is an acute demyelinating polyradiculoneuropathy rare in pediatrics. The association between confirmed viral hepatitis A and GBS is extremely rare; 18 cases which are described in the literature.

Objective of the Work: Emphasize this rare association as well as add another case to the 18 cases described in the literature through an observation collected at the pediatrics A department of CHU Mohamed VI in Marrakech and review of the literature.

Observation: He is a 9-year-old child from a non-consanguineous marriage with a history of hepatitis A confirmed by the positivity of the antibody against hepatitis A type IgM; occurred 8 days before symptomatology.

The patient was admitted for ascending quadriplegia associated with swallowing disorders and respiratory distress. The initial clinical examination objectified a peripheral motor deficit with abolished ROTs and major respiratory distress. The child was initially admitted to the intensive care unit or was intubated and tracheotomized. The lumbar puncture had objectified an albumino-cytological dissociation and the EMG was in favor of a severe acute polyradiculoneuritis.

The management consisted in the administration of intravenous immunoglobulins as well as 5 plasma exchange sessions.

The recovery phase was very gradual with almost complete healing thanks to motor physiotherapy.

Conclusion: The association of hepatitis A and GBS is extremely rare in the pediatric population and incites the vigilance of doctors before any neurological sign occurring during the surveillance of infections by viral hepatitis A.

Keywords: *Guillain-Barré syndrome (GBS); Polyradiculoneuropathy; Hepatitis A*

Introduction

Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyradiculoneuropathy. It is a rare affection in childrens responsible for neurological symptomatology secondary to demyelination of peripheral neurons. Some etiological explications are now well known and others are suspected. In general, are viral or bacterial antigens with sometimes a previous clinical infectious episode.

The combination of hepatitis A and SGB is described in 18 cases in the literature. Our observation is a new case that is added, report the occurrence of GBS after hepatitis A infection with hepatitis A.

Case Report

A 9-year-old child from a first-degree consanguineous marriage consulted in the emergency department for respiratory and walking disorders that appeared gradually over two days. His notable medical history was hepatitis A occurred 8 days before the symptomatology; suspected in asthenia, anorexia, cholestatic jaundice and hepatic cytolysis (ASAT: 1170 and ASAT: 1330) and confirmed by the positivity of the antibody against hepatitis A IgM type.

The patient initially presented paresthesia of the lower limbs complicated by a symmetric and ascending motor deficit in both upper limbs. The evolution was marked by the installation 24 hours after deglutition disorders and respiratory distress.

The patient was initially admitted to the pediatric intensive care unit. The initial examination showed a sleepy, polyphonic child at 35 cycles per minute, normotensive at 09/05 mmHg and apyrexis. The neurological examination found a predominantly proximal tetraparesis with muscular deficit of the pelvic and scapular belts rated at 2/5 associated with a distal motor deficit of 3/5. The osteotendinous reflexes were abolished. The deep sensibility was diminished. He had major respiratory distress. The rest of the clinical examination did not find any abnormalities, in particular no hepatomegaly.

The initial lumbar puncture had demonstrated albumin-cytological dissociation with 3-cell cellularity and 0.77 protein-choline.

An electromyogram was performed showing motor and sensory absence of potential to the various nerves and confirming the diagnosis of severe acute polyradiculoneuropathy. The first biological results did not find any infectious syndrome, inflammatory syndrome or ionogram disturbance. Hepatic cytolysis was in decline (ASAT 311 and ALT 394).

The child was intubated initially ventilated and tracheotomized on the 2nd day of admission. Intravenous immunoglobulin treatment was immediately undertaken for a period of 2 days.

The evolution was marked by a deterioration of the neurological and respiratory state requiring 5 sessions of plasma exchanges.

The plateau phase lasted 30 days with stabilization of the motor deficits. The child also had neuropathic pain that was put on Gabapentin and Amitriptyline.

The recovery phase was very progressive with almost complete improvement thanks to motor physiotherapy.

The significant sequelae during the surveillance are a heaviness of the right lower limb still under motor rehabilitation with a decline of 10 months.

Discussion

GBS is the most common cause of acute peripheral paralysis in children after the eradication of acute poliomyelitis in Morocco.

All children can be affected with a significant prevalence between 4 and 6 years old and it is exceptional in the neonatal period [1].

It is an acute demyelinating polyradiculoneuropathy that presents itself in its typical form by a motor neurological involvement that is both motor and sensory. Fulminant forms are possible with a rapid evolution in 48 hours, installation of quadriplegia and use of mechanical ventilation [2].

The abolition of osteotendinous reflexes is an early sign. Sensory disturbances such as paresthesia or numbness are very frequently associated with deficit signs.

The albumino-cytological dissociation of cerebrospinal fluid is a major but not obligatory criterion. The hyperproteinorrachia is greater than 0.45 g/l to more than 10 g/l with no cellular reaction or a slight pleocytosis of 20 to 50 elements/mm^3. Hyponatraemia by inappropriate secretion of antidiuretic hormone is also reported but may be secondary to treatment with intravenous immunoglobulin [3].

The EMG makes it possible to establish the diagnosis as well as to establish the progressive prognosis.

GBS is the consequence of an abnormal immune response with the production of antibodies directed against the myelin of peripheral nerves following an attack most often viral; Some external antigens are strongly suspected but often difficult to demonstrate in clinical practice.

Viruses of the family Herpes are classically one of the viruses most often described [4].

Acute viral hepatitis B, acute hepatitis C, hepatitis D, hepatitis E are more described responsible for GBS and rarely hepatitis A [5].

Hepatitis A infection elicits an immune response, which in turn cross-reacts with peripheral nerve components due to sharing of cross-reactive epitopes (molecular mimicry) [6].

The association viral hepatitis A and SGB was first described in 1929 [7]. An outbreak of viral hepatitis A occurred in Shanghai between January and March 1988, and of the 292,301 cases of HA 8 had GBS [8].

A review of the literature revealed only 18 cases of serologically confirmed viral hepatitis A associated with GBS; of whom 4 were children [9,10]. The onset of neurological signs begins between 7 days and 15 days after infection with viral hepatitis A. In most cases, only 1 patient presented GBS parallel to the infection and 1 case after 21 days and the last case published after 5 weeks [9,10].

GBS associated with the hepatitis A virus is associated with male preponderance, early age and better prognosis. Fulminant hepatitis A does not appear to correlate with the severity of neurological symptoms [11].

The association between GBS and hepatitis A infection could be a marker of good prognosis unlike *C. jejuni* [11].

Our child meets all the clinical, biological and electrical criteria for the diagnosis of GBS. Chronologically, the occurrence of neurological signs was early compared to other cases reported in the literature. Our patient presented a non-standard form called fulminant GBS with rapid installation of quadriplegia and respiratory failure. The management was early and the favorable evolution joined the literature.

This observation leads us to reinforce the hypothesis of the association of the GBS and the infection by the hepatitis A virus. Our child is a new case which is added to the 4 other children previously published.

Conclusion

The combination of SGB and viral hepatitis A is rare and especially in the pediatric population, which may be responsible for all forms of GBS with a variable delay.

This association encourages the interest of the vigilance of the doctors in front of any neurological sign occurring during the surveillance of the infections by the viral hepatitis A.

Conflict of Interest

No conflict of interest.

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