Short Communication

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In Juvenile Idiopathic Arthritis, Immunisation and Vaccine Safety

Abstract

Background

Autoimmune mechanisms and medicines used in treatment increase the threat of liver complaint in cases with juvenile idiopathic arthritis (JIA) and hepatitis A contagion (HAV) vaccination is important, especially in intermediate- endemicity areas like Turkey. In our study, we aimed to estimate the vulnerable response to hepatitis A vaccine and vaccine safety in children with JIA.

Methods

This study was carried out in our sanitarium's Pediatric Rheumatology inpatient clinic and Healthy Child clinic between the times 2003 and 2008. The study group comported of 47 children with JIA (23 joker and 24 womanish) diagnosed according to International League of Associations for Rheumatology individual criteria. The control group comported of 67 healthy children (31 ladies, 36 manly) who didn't have a history of hepatitis A infection or vaccination. Both groups were vaccinated with two boluses of hepatitis A vaccine at 6- month intervals. Anti-HAV IgG> 80 MIU was accepted as positive response.

Results

There was no significant difference between the groups in terms of age and coitus. None of the cases with JIA had fever, clinical worsening, or complaint activation after vaccination. Anti-HAV IgG positivity rate was significantly advanced in the control group (p<0.05). Anti-HAV IgG was negative in only four cases, and they were all manly cases with systemic JIA who had active complaint underanti-tumor necrosis factor treatment.

Conclusion

Hepatitis A vaccine was safe in cases with JIA, and response to vaccine didn't differ between healthy children and cases with JIA except for children with active systemic JIA enteringanti-tumor necrosis factor nascence medicines.

Keywords: Anti-hepatitis A IgG • Anti-tumor necrosis factor medicines • Hepatitis A vaccination • Juvenile idiopathic arthritis

Introduction

Systemic lupus erythematosus (SLE) is an autoimmune rheumatic complaint that generally affects women during travail period. SLE flares have been linked to reproductive hormones. Prolactin (PRL) acts as a hormone due to pituitary stashing; and as a cytokine due to extrapituitary product. Utmost of the vulnerable cells cache PRL which stimulate proliferation, isolation and development of T and B lymphocytes. It amplifies interleukin- 2(IL- 2) action and inhibits lymphocytes apoptosis. PRL, as an important immunomodulator, is linked with a number of rheumatic and autoimmune conditions [1]. It was intertwined in the pathogenesis of SLE. Hyperprolactinemia (hPRL) was observed in SLE cases of both genders and was associated with complaint exertion. Still, some studies didn't support this association. The part of prolactin in the pathogenesis of SLE isn't yet conclusive. There's failure of data on PRL and its relation to SLE from Arab countries. The

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Material and Methods

This study was carried out in our sanitarium's Pediatric Rheumatology inpatient clinic and Healthy Child clinic between the times 2003 and 2008. The study group (Group 1) comported of 47 children with JIA(23 joker and 24 womanish) diagnosed according to International League of Associations for Rheumatology individual criteria that were edited after the Edmonton Arrangement in2001.6 The parents were questioned about vaccination of their children against hepatitis A and hepatitis infection memory. Hepatitis A antibodies(anti-HAV IgM and IgG) were studied by macro enzyme- linked immunosorbent assay with Macro Access bus- analyzer in all of the children with no memory of vaccination and hepatitis A infection [3].7Anti-HAV IgG> 80 MIU was accepted as positive response. Children in who Manti-HAV IgG was negative were included in the study. None of the children hadanti-HAV IgM positivity. Children with habitual complaint other than JIA and children who didn't regularly come to follow- up were barred. The control group(Group 2) comported of aimlessly named 67 healthy children(31 lady, 36 manly) with analogous age and coitus as the study group who applied to our inpatient clinic for several health instrument or webbing tests and who didn't have a history of hepatitis A infection or vaccination. Anti-HAV IgM and IgG were estimated in all of the children. Children with negativeanti-HAV IgG were included in the study [4]. None of the children hadanti-HAV IgM positivity. Complaint exertion was estimated with the Childhood Health Assessment Questionnaire (CHAQ).8 Children who didn't have any complaints in the last 6 months with CHAQ scores lower than0.5 were accepted as being in absolution. Children who had symptoms of JIA in the last 6 months and children who had to take fresh medicines with CHAQ scores advanced than0.5 were accepted as being in active phase. Informed concurrence was attained from the parents of all of the children. The study was approved by the ethics commission of the sanitarium. Both groups were vaccinated with two boluses of hepatitis A vaccine at 6- month intervals. Anti-HAV IgG titters were estimated at an normal of 2 months after the alternate cure of hepatitis A vaccine [5].

Results

This study was carried out between October 2003 and October 2008 in our sanitarium's Pediatric Rheumatology inpatient clinic and Healthy Child clinic. The study group comported of 47 cases(24 lady, 23 joker) with JIA with a mean age of 10.73 ± 3.89 times, and the control group comported of 67 healthy children(31 lady, 36 manly) with a mean age of 9.41 ± 3.80 times [6].

Discussion

HAV is the most common cause of hepatitis in nonage and an important public health problem, especially in intermediate-aboriginal areas like our country.9 It's also the most common cause of fulminant hepatic failure in Turkish children10 as well as in numerous other countries around the world. According to the World Health Organization, roughly1.5 million clinical cases of hepatitis A do worldwide annually, 1 but seroprevalence data indicate that knockouts of millions of HAV infections do each time. In areas of moderate endemicity, HAV isn't transmitted as readily because of better aseptic and living conditions, and the average age of infection is advanced in these areas than in areas of high endemicity [7]. Paradoxically, the eventuality for large outbreaks of hepatitis A can be increased in comparison with largely aboriginal areas, because there's a larger pool of susceptible aged children and grownups(compared with high- endemicity countries) who are at high threat of infection and who, when infected with HAV, are likely to develop characteristic illness.14 The stylish way of controlling HAV endemicity is vaccination. Hepatitis A vaccines presently certified are prepared from inactivated HAV and are original in terms of immunogenicity and efficacity; still, some medical conditions that beget immunosuppression might reduce vulnerable response [8].

Vaccination is an important process in children and adolescents with seditious rheumatologic complaint. Active infection can lead to severe problems especially in immunosuppressed cases. Russo etal. Reported two JIA cases that developed macrophage activation pattern after hepatitis A infection.15 One of them entered absolution after high- cure steroids and immunosuppressant's, and the other failed. In another case from England, 20- time-old womanish with Still's complaint who had hepatitis A was diagnosed with contagion- associated hemophagocytic lymphohistiocytosis, 16 and one case from China with systemic- onset JIA developed macrophage activation pattern after hepatitis A infection [9].

Conclusion

We can say that hepatitis A vaccine is safe and immunogenic in cases with JIA, and response to vaccine didn't differ between healthy children and cases with JIA

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except for children with active systemic JIA enteringanti-TNF nascence medicines [10].

Conflict of Interest

None

Acknowledgment

None

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