OPEN ACCESS Freely available online

Pediatrics & Therapeutics

ISSN: 2161-0665

Case Report

Herpes Simplex Virus Type-1 Encephalitis in an Infant with Kawasaki Disease

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ABSTRACT

Kawasaki disease is an acute febrile vasculitis in children less than 5 years old. Infection is probably considered as a predisposing factor with complications such as aseptic meningitis, encephalitis, seizures, and irritability. We report a case of a 3-month-old boy with Kawasaki Disease (KD) complicated with HSV type-1 encephalitis.

Key words: Kawasaki disease; Neonatal encephalitis; Herpes simplex virus type-1

INTRODUCTION

Kawasaki Disease (KD) is an acute febrile systemic vasculitis of childhood. The disease usually occurs in children less than 5 years old, with greater prevalence in the Asian population. The cause and pathogenesis of KD is still not completely known. Systemic vasculitis in KD can result in multisystem organ involvement including the Central Nervous System (CNS) with complications such as aseptic meningitis, encephalitis, seizures, ataxia, and irritability. Viral infections like Epstein–Barr Virus (EBV), Cytomegalovirus, Herpes Simplex Virus (HSV), Varicella Zoster Virus (VZV), Human herpes-virus 6, Influenza, Human immunodeficiency virus, Dengue Virus (DV) are most frequent associated with KD [1]. We report a case of a 3-months-old boy with KD complicated with HSV type-1 encephalitis.

CASE PRESENTATION

A three-month-old baby boy with a normal birth history, growth and development history presented with a high-grade fever of 2 days duration. He was drowsy and lethargic. Detailed physical examination not revealed any maculopapular rash, conjunctivitis, or any other associated symptoms or signs such as rhinitis, cough, difficulty in breathing, rash, diarrhoea, vomiting, abdominal distention, fissured lips, and cervical lymphadenopathy. He had a fever of 39°C. Meningeal signs were negative. His cardiac parameters were also normal. He did not have a contact history of fevers or any other infectious illnesses. Routine blood investigations revealed marginal leukocytosis, normal platelets, marginally elevated with normal AST, and slightly elevated C-Reactive Protein (CRP).

His bacterial infection screen compromising of blood, urine, sputum, and CSF cultures were negative. The viruses tested for were dengue NS1 antigen in blood, JE IgM in blood and CSF, HSV 1 and 2, VZV in CSF with alpha herpes virus real time PCR, and enterovirus in CSF with real time PCR. All were negative except for HSV type -1 which became positive in CSF with a real time Polymerase Chain Reaction (PCR) (Figure 1).

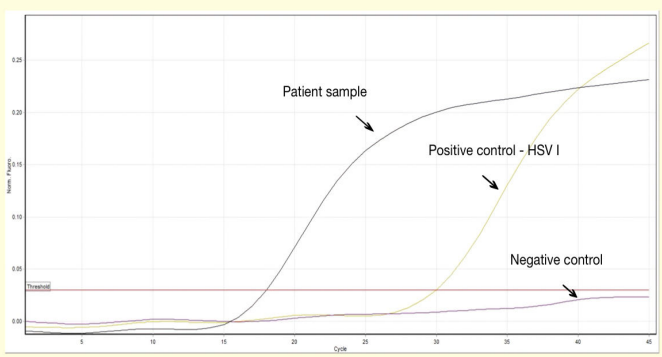

Figure 1: The qualitative qPCR analysis of the baby for HSV-1.

On further inquiry, the ward informed that the drowsiness and lethargy had resolved on the subsequent day after admission and the patient was later diagnosed to have KD. He was thus being treated for KD when results of HSV-1 became available. IV acyclovir had not been initiated on admission. However, IV acyclovir was initiated at the time of HSV-1 positive and only continued for 3 days since the patient was free of CNS symptoms very early days of hospitalization with an alternative diagnosis.

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Received: 19-Jan-2024, Manuscript No. PTCR-24-29185; **Editor assigned:** 22-Jan-2024, Pre QC No. PTCR-24-29185 (PQ); **Reviewed:** 05-Feb-2024, QC No. PTCR-24-29185; **Revised:** 12-Feb-2024, Manuscript No. PTCR-24-29185 (R); **Published:** 19-Feb-2024, DOI:10.35841/2161-0665.24.14.539.

Citation: Abeynayake JI, Mahanama AIK, Fernando MAY, Weligamage DDCU (2024) Herpes Simplex Virus Type-1 Encephalitis in an Infant with Kawasaki Disease. Pediatr Ther. 14:539.

RESULTS AND DISCUSSION

Encephalitis due to HSV (HSE) can cause significant morbidity and mortality among all age groups. HSV type-2 is the commonest organism implicated in neonates and infants up to about 3 months of age, while in children and adults, the predominant agent is HSV type-1[2]. Neonates almost always acquire HSV type-2 during the perinatal period following exposure to maternal asymptomatic or symptomatic (genital or occasionally oral lesions) infection [3].

Though neonatal HSE due to HSV-1 is uncommon, they also occur in about 10% of cases following exposure to a person with primary manifestations such as finger infection, oral lesions, or other cutaneous lesions during the post-natal period. Hence, HSV-1 infection in this patient may have occurred following a post-natal exposure. Lack of preceding history of skin and cutaneous lesions makes it difficult to identify a source which is also not uncommon for HSE.

Definitive diagnosis of HSE is mainly dependent on molecular demonstration of HSV1 and 2 DNA in CSF, as occurred in this patient. CSF had been collected during the initial period of the presentation, and HSV1 and 2 type detection real time PCR (a commercially validated assay) gave a positive result with a significant CT value indicating the presence of a high viral concentration within the CSF. Regardless of the type of HSV identified management of the patient remains the same [4,5] which is initiation of IV acyclovir as early as possible (within 4 hours) on suspicion and continuation up to 21 days or longer depending on response. However, in this patient IV acyclovir had not been initiated at the presentation and later a very short course of IV acyclovir was administered due to HSV-1 positivity in CSF. However, it was discontinued due to the change of the clinical scenario leading to a diagnosis of KD. A repeat CSF was not tested to demonstrate the clearance of the virus from CSF, because the child had recovered from encephalitic symptoms when the test results were made available. Uneventful recovery from HSE may be explainable from certain literature which states that HSV-1 encephalitis has a better prognosis than HSV-2 during neonatal period [2,3,4].

Had HSV-1 acted as a predisposing factor for KD?

KD is an acute febrile vasculitic condition affecting mainly the medium sized arteries in children during their early childhood (usually less than 5 years of age). It is usually self-limiting, however its predilection for the coronary arteries can lead to development of coronary artery aneurysms leading to sudden death. Complications due to CAA can be minimized by early identification and treatment (risk of CAA 25% in the untreated to 3-5% in the treated). Thus, a high degree of suspicion for early identification and prompt treatment (initiation of Intravenous immune globulin (IVIG) within 10 days after the onset of fever and therapy with aspirin) is important for better prognosis.

The incidence of KD is very much higher in the Asian population than that of USA as compared to 250/100,000 children <5 years of age in japan to 25/100,000 children less than 5 years in the USA. Diagnosis of KD is mainly clinical. Complete KD is diagnosed based on the presence of fever for more than 5 days along with 4-5 of the principal clinical features such as extremity changes, polymorphorious rash, oropharyngeal changes, bilateral non-exudative bulbar conjunctival injections and acute unilateral non purulent cervical lymphadenopathy (diameter greater than 1.5 cm) [6].

The etiology for KD is yet to be defined and infections are considered to be an important predisposing factor. At the same time autoimmune conditions and genetic factors have also been suggested. Among infectious agents both bacterial (*Neisseria meningitides*, Bacterial toxin-mediated super antigens, *Mycoplasma pneumonia* and *Klebsiella pneumonia*) and viral agents have been suggested. Most commonly implicated viruses are Parvo B19, Adenovirus, Cytomegalovirus (CMV), Parainfluenza-3, Rotavirus, EBV, HTLV, Measles, etc., [1]. Moreover, a so-called KD agent which is of viral etiology characterized by intracytoplasmic inclusion bodies, has also been found to be associated with KD.

CONCLUSION

The relationship between the HSV-1 infection and the KD later diagnosed in this patient remains unclear. Though CMV, EBV, HHV-6 like herpes viruses have been suggested as possible predisposing viruses, report of KD due to HSV-1 and 2 are scares. In this patient, HSV-1 infection may have acted as a separate entity giving rise to encephalitis which may have had a benign cause culminating in spontaneous recovery. Moreover, a case report of KD following HSV-1 positivity in CSF was published in the Journal of Babol University of Medical Sciences-a rare case of KD following the herpetic encephalitis type-1 by Khodabandeh M et al., [7] describing the importance of having a high index of suspicion for both diseases in patients presenting with similar symptoms, for early diagnosis and management for better outcome. This case also emphasizes the importance of careful evaluation of laboratory results and consideration of all clinical clues before embarking on specific therapy in order to provide best patient care.

AUTHOR CONTRIBUTIONS

A.I.K. Mahanama-original draft of the manuscript, M.A.Y. Fernando-laboratory diagnostic procedures, D.C. U. D. Waligamage-review and editing the manuscript, J.I. Abeynayake-expert opinion on laboratory diagnosis and review the final manuscript.

ACKNOWLEDGMENT

We acknowledge the staff the Department of Virology, Medical Research Institute, Colombo.

DECLARATION OF COMPETING INTEREST

The authors declare that they have no competing interests.

DATA AVAILABLE STATEMENT

Data sharing is not applicable to this article as no data sets were generated or analyzed during the current study.

ETHICS STATEMENT

This short report includes the clinical information of an individual patient. However, no patient-identifying details were mentioned.

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