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Case Report

Cytomegalovirus Induced Neonatal Cholestasis: A success Story

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Abstract

Neonatal cholestasis is a prolonged elevation of the serum levels of conjugated bilirubin beyond the first 14 days of life. Commonest etiologies include extrahepatic conditions like biliary atresia, intrahepatic like congenital malformations, infections and inborn errors of metabolism. TORCH infections constitute 22% cases of neonatal hepatitis, of which Cytomegalovirus is the commonest agent. Ganciclovir and its prodrug valganciclovir are recommended in the management of Cytomegalovirus infection. We, hereby report a 2 month old child, a case of Cytomegalovirus induced neonatal cholestasis with marked improvement in clinical and biochemical parameters, post treatment with intravenous ganciclovir.

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Keywords

- Neonatal cholestasis
- Cytomegalovirus
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INTRODUCTION

Cholestatic jaundice affects approximately 1 in every 2,500 infants [1,2]. It is estimated that approximately 40% of cholestasis in infants is due to neonatal hepatitis [3]. Human cytomegalovirus (CMV) is 1 of 8 human herpes viruses [4], which is the commonest etiological agent responsible for causing neonatal hepatitis [5]. Ganciclovir was the first antiviral agent approved for the treatment of CMV infection. In the past few years, there have been various trials based on the outcome of ganciclovir therapy in CMV induced neonatal hepatitis with satisfactory results [3,4,6]. This case report highlights the outcome of ganciclovir treated CMV neonatal hepatitis.

CASE REPORT

A two month old female child born of non-consanguineous marriage presented with yellowish discoloration of eyes and skin since day two of birth. There was history of passing clay colored stools since past 1 month. The child was born full term with birth weight of 3 kgs. Antenatal history revealed that the mother was a case of hypothyroidism, on anti-thyroid medication since past two years and HIV status was non- reactive. The child was developmentally normal and belonged to a lower middle class family.

On general examination, her weight was 4.4 kgs; length was 54cms, with pallor and icterus. On her abdomen examination there was hepatosplenomegaly (Figure 1) with the rest of the systems being normal.

Investigations showed Hemoglobin of 11.6gm/dl, white cell count of 24,800/cumm (35% polymorphs, 60% lymphocytes) with platelet of 461,000/cumm. Liver function showed evidence of direct hyperbilirubinemia with total bilirubin of 23.6 mg/dl, direct bilirubin of 16.2 mg/dl, alanine transaminase=353IU/L, aspartate transaminase= 850 IU/L and alkaline phosphatase =1156 IU/L. Prothrombin time, partial thromboplastin time, serum electrolytes, blood gases and serum ammonia were within normal limits. Thyroid profile and Glucose-6-phosphate dehydrogenase tests were normal. Ultrasonography of the abdomen reported hepatosplenomegaly with normal gall bladder. Hepatobiliary Scintigram after five days of priming with phenobarbitone showed hepatosplenomegaly with moderate impairment of parenchymal function of the liver without any evidence of biliary atresia. TORCH screening showed positive CMV-IgM titer of 6.8 UA/ml (reactive >2.0 UA/ml).An ophthalmic and hearing evaluation was done which was normal. Liver biopsy could not be performed due to negative consent given by the parents for the procedure. The child was then started

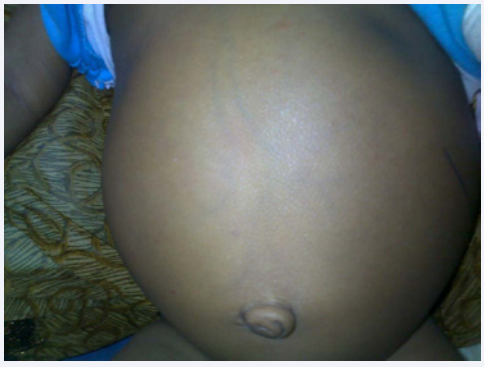

Figure 1 Showing abdominal distention due to hepatosplenomegaly.

Figure 2 Child on follow-up.

on intravenous ganciclovir (10mg/kg/day) for 21 days with close monitoring of the complete blood counts and renal function tests. She was also given fat soluble vitamins along with Ursodeoxycholicacid (UDCA) supplements. Post therapy, a decrease in total and direct bilirubin, liver transaminases and CMV IgM titers along with regression of hepatosplenomegaly was noted. At 6 months of age, a repeat Hepatobiliary Scintigram showed minimal impairment of parenchymal liver function. A regular growth and development assessment was done every 6 months post treatment. Currently the child is three years old with normal developmental milestones, gaining weight and height adequately for her age (Figure 2).

DISCUSSION

Recently there have been studies supporting the use of ganciclovir to treat CMV induced neonatal hepatitis leading to neonatal cholestasis. Anti-viral treatment is valuable in cases of intrahepatic cholestasis due to CMV after ruling out other causes [5]. The liver function tests and direct bilirubin in patients with Cholestatic jaundice have improved post treatment with this drug [5]. Ganciclovir is a synthetic nucleoside analogue 2-deoxyguanine which inhibits the cytomegalovirus replication [4]. Its common side effects are bone marrow suppression resulting in pancytopenia, nephrotoxicity, rash, vomiting and nausea [4]. Literature review has shown clinical trials supporting the use of ganciclovir and its oral prodrug valganciclovir in the treatment of CMV induced neonatal cholestasis. Xavier et al and Fischler et al have reported good treatment outcome with use of ganciclovir in such cases [5,7]. Similarly, the child in our reported case was treated with ganciclovir for 3 weeks under close monitoring, during which no side effects were observed. There was significant improvement in child's clinical condition post anti-viral treatment. This case report warrants the need for guidelines based on clinical trials for the use of ganciclovir in such cases.

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