

Correlation of Hepatitis C Virus Infection with Multiple Blood Transfusions in Thalassemic Children

Saurabh Chhotatal Gamit¹, Pushpa Ramjibhai Kateshiya^{2*}, Dipali Maganbhai Gavali³

¹Department of Microbiology, Smt. B. K. Shah Medical Institute and Research Centre, Sumandeep Vidyapeeth Deemed to be University, Piparia, Vadodara, Gujarat, India

²Department of Microbiology, Shri. M. P. Shah Govt. Medical College, Jamnagar, Gujarat, India

³Department of Microbiology, Parul Institute of Medical Sciences and Research, Limda, Vadodara, Gujarat, India

ABSTRACT

INTRODUCTION

Thalassemia is blood disorder passed down through families caused in which the body makes an abnormal form or inadequate amount of hemoglobin. Hemoglobin is the protein in RBCs that carries oxygen, leading to hemolytic anemia. Hemolytic anemia in patients with thalassemia necessitates repeated blood transfusions. Multiple transfusions are associated with complications, such as hepatitis B and C and certain sexually transmitted disease like, HIV infections, syphilis infection and iron can build up in tissues. This study aimed to determine the prevalence of hepatitis C in children with multi-transfused thalassemia.

MATERIALS AND METHODS

This study was carried at the Department of Microbiology, Shree Meghaji Pethraj Shah Medical College and Guru Govind Govt. Hospital, Jamnagar from January 2017 to December 2018. Patients with thalassemia who received multiple blood transfusions were included in this study.

RESULTS

Serum samples from 227 patients are, including 134(59.1%) boys and 90(40.9%) girls, were tested using enzyme-linked immune sorbent assay kits for hepatitis C. Of them 38(16.7%) were reactive for hepatitis C virus antibodies.

CONCLUSION

Sensitive screening tests, strict guidelines for donor selection before blood transfusion in thalassemia management are needed for prevention of transfusion related infections among thalassemic patients.

KEYWORDS

Hepatitis C virus, Thalassemia, ELISA, Blood transfusion

** Corresponding Author:*

*Pushpa Ramjibhai Kateshiya,
Department of Microbiology, Shri. M.
P. Shah Govt. Medical College,
Jamnagar, Gujarat, India;
E-mail:
pushpa.22kateshiya@gmail.com*

How to Cite This Article:

*Gamit SC, Kateshiya PR, Gavali DM.
Correlation of Hepatitis C Virus
Infection with Multiple Blood
Transfusions in Thalassemic Children.
J Evid Based Med Health
2023;10(2):87.*

*Received: 05-Jul-2023;
Manuscript No: JEBMH-23-104962;
Editor assigned: 07-Jul-2023;
PreQC No. JEBMH-23-104962(PQ);
Reviewed: 21-Jul-2023;
QC No. JEBMH-23-104962;
Revised: 28-Jul-2023;
Manuscript No. JEBMH-23-104962(R);
Published: 07-Aug-2023;
DOI: 10.18410/jebmh/2023/10/02/87*

*Copyright © 2023 Gamit SC, et al.
This is an open access article
distributed under Creative Commons
Attribution License [Attribution 4.0
International (CC BY 4.0)]*

INTRODUCTION

Hepatitis C Virus (HCV) falls in to the genus *Hepacivirus*, a member of the family Flaviviridae, and is a single stranded RNA virus. An estimated 180 million people are affected by HCV infection, with a worldwide prevalence of 2%, accounting for 3 million people every year [1]. HCV is mostly transmitted through the prenatal route, thereby making it a common complication of multiple blood transfusions. It enters liver cells and causes severe inflammation in the liver [2]. HCV infection chronically causes cirrhosis and hepatocellular carcinoma [3,4]. During 2010–2019, 1.83 million people \leq 65 years of age died from HCV infection [5]. As per study done by WHO, 170 million people are affected by HCV all over the world [6].

Thalassemia is blood disorder passed down through families caused in which the body makes an abnormal form or inadequate amount of hemoglobin. Hemoglobin is the protein in RBCs that carries oxygen, leading to hemolytic anemia. Patients with thalassemia develop mild or severe anemia. Severe anemia can organ damage and lead to death. α -thalassemias results from HBA1 and HBA2 mutations, due to this reduction of alfa globin chain effectively, leading to fewer α -globin chains with excess beta chains in adults and gama chains in newborns. β -thalassemias results from HBB mutations on chromosome 11, passed down through families recessively, resulting in reduction or absent beta-chain [7]. Treatment of thalassemia major mainly involves repeated blood transfusions at several intervals, which can lead to iron can build up in tissues. Excess of iron in tissues in thalassemic patients can be treated with chelation therapy with EDTA. More severe forms of thalassemia, such as severe anemia lasting over 2 weeks, often require frequent blood transfusions, possibly every few weeks. By this we can reduce growth deficiency, organ destruction and bone impairment, and improving activities of daily living and quality of life [8,9]. Multiple transfusions are associated with complications, such as hepatitis B and C and certain sexually transmitted disease like, HIV infections, syphilis infection and iron can build up in tissues [10]. The β thalassemia disorders pose a significant health burden in India. The average prevalence of β thalassemia carriers is 3%–4% which translates to 35 to 45 million carriers in our multi-ethnic and culturally and linguistically diverse population of 1.21 billion people which also includes around 8% of tribal groups according to the Census of India 2011. Several ethnic groups have a much higher prevalence (4%–17%), 7%–25% of who develop HCV infection due to multiple transfusions [11–13]. Present study done for to find out the frequency of HCV infection and risk factors in children with multi-transfused thalassemia to establish preventive strategies and investigate the efficacy of routine screen methods for the find out HCV infected donor.

MATERIALS AND METHODS

This study was carried at the Department of Microbiology, Shree Meghaji Pethraj Shah Medical College and Guru Govind Govt. hospital, Jamnagar, from January 2017 to December 2018. During the study, serum samples were collected from 227 patients with thalassemia who received multiple blood transfusions.

All needed history of thalassemic children taken by detailed interviewing of the patient and/or there relative such as age,

duration and how many time they received blood. Total 5 ml of blood sample was withdrawn by venipuncture using sterile disposable syringes by aseptic precautions of each patient after that blood inject in to the clot activator red vacutue. Then this whole blood was rotate by 10000 rpm for 5 min for serum separation. After that clear serum was transferred into sterile vials by using pipette. After that an ELISA test done for qualitative detection of antibodies to hepatitis C virus (Anti-HCV) in human serum as per kit manual. The kit used in testing was Qualisa 3rd generation enzyme linked immunosorbent assay for detection of antibody to HCV in human serum which was manual ELISA method. The absorbance value was taken in ELISA reader at 450 nm with 600 nm–700 nm as reference, after that cut-off value was calculated by adding 0.3 to average absorbance value of negative control ($COV = Av.NC + 0.3$) and interpreted result as per manufacturer's kit inserts if sample with absorbance value less than cut-off value considered non-reactive and more than cut-off value considered reactive for HCV antibodies. Initially reactive sample retested in duplicate, if these samples that do not react in either of duplicates are consider negative. If sample is repeatedly reactive in either of duplicated are consider repeatedly reactive.

RESULTS

227 thalassemic children were included in this study, out of these 134 (59.1%) boys and 93 (40.9%) girls. Of them, 38(16.74%) were positive for anti-HCV antibodies. The HCV seroprevalence in boys and girls was 15.67% and 18.27%, respectively. Among HCV-positive patients with thalassemia, the majority cases were found in 9–12 years age group at 17(44.74%) cases. The majority cases of hepatitis C virus infection detected in children who received \geq 100 blood transfusions (52.94%), while the lowest was seen in patients with fewer than 25 transfusions, indicating that the hepatitis C virus infection was directly proportionate with the number of blood transfusions (Tables 1–3).

| | No. of patients (n=227) | Reactive to anti-HCV antibodies (n=38) |
|--------|----------------------------|---|
| Male | 134 | 21 (55.3%) |
| Female | 93 | 17 (44.7%) |
| Total | 227 | 38 (16.74%) |

Table 1. Sex Distribution of the Prevalence of Anti Hepatitis C Virus Antibodies in Children with Beta Thalassemia.

| Age (years) | No. of patients (n=227) | Reactive to anti-HCV antibodies (n = 38) |
|----------------|----------------------------|---|
| <2 | 12 | 1 (8.33%) |
| 2–4 | 41 | 1 (2.43%) |
| 5–8 | 60 | 5 (8.33%) |
| 9–12 | 45 | 17 (37.78%) |
| 12–18 | 69 | 14 (20.29%) |

Table 2. Age Distribution of the Prevalence of Anti-Hepatitis C Virus Antibodies in Children with Beta Thalassemia.

| Number of transfusions | No. of patients (n=227) | Reactive to anti-HCV antibodies (n=38) |
|------------------------|-------------------------|--|
| 0-25 | 77 | 3 (3.9%) |
| 26-50 | 68 | 06 (8.8%) |
| 51-75 | 33 | 09 (27.3%) |
| 76-100 | 30 | 11 (36.7%) |
| >100 | 17 | 09 (52.9%) |

Table 3: Association of the Number of Blood Transfusions with Hepatitis C Virus Infection in Children with Beta Thalassemia.

DISCUSSION

HCV infection in Indian children with thalassemia showed a prevalence rate between 7.8% and 60%^[14]. The prevalence of HCV infection in present study was 16.74% similar to Modi et al., Agarwal et al. and Mukharjee et al. studies (Table 4)^[15-16].

| Study | Hepatitis C virus infection | Male | Female |
|----------------------------------|-----------------------------|--------|--------|
| Present study | 16.74% | 55.30% | 44.70% |
| Mukherjee et al. ^[14] | 24.64% | - | - |
| Modi et al. ^[15] | 20.40% | 68.40% | 31.60% |
| Agrawal et al. ^[17] | 24.00% | - | - |

Table 4. Comparison of the Seroprevalence of Hepatitis C Virus Infection in Children with Beta Thalassemia between the Present and Previous Studies.

In previous studies involving children with thalassemia, HCV infection showed no significant difference between boys and girls while showing a positive correlation with the number of blood transfusions. The risk of acquiring HCV infection increases with an increased number of transfusions which is supported by Surani et al. Modi et al., and Agarwal et al. studies (Table 5)^[15-16].

| Number of transfusions | Present study | Chandani et al. ^[15] | Modi D et al. ^[14] | Agrawal et al. ^[16] |
|------------------------|---------------|---------------------------------|-------------------------------|--------------------------------|
| 0-25 | 3.89% | - | 14.60% | 10.70% |
| 26-50 | 8.82% | - | | |
| 51-75 | 27.27% | 7.69% | 9.50% | 15.50% |
| 76-100 | 36.66% | 18.18% | | |
| >100 | 52.94% | 40.00% | 35.60% | 28.00% |

Table 5. Association of the Number of Blood Transfusions with Hepatitis C Virus Infection in Children With Beta Thalassemia in the Present and Previous Studies.

CONCLUSION

In our study, HCV infection showed no sex predilection in children with multi-transfused thalassemia. The incidence of HCV infection tended to increase with the number of blood transfusions. The major reasons for transmission are the non-availability of vaccines against HCV. Patients with thalassemia may be infected during the transfusion of hepatitis C virus infected blood during the donor window period. The only way to prevent HCV infection in patients with multi-transfused thalassemia is by establishing standard and cost-effective testing methods other than ELISA techniques and effective guidelines for screening of blood and early detection of HCV infection, particularly in the window period. Pre-donation screening of blood donors for Transfusion Transmissible Infections (TTI) is the practice by which a prospective donor is tested for the presence of HCV infection a single rapid or quick method, and donation is deferred if the test is reactive for HCV antibody.

ACKNOWLEDGEMENT

The authors are thankful to the institution for granting permission to conduct the study and to the teaching staff of the Department of Microbiology, Shree Meghaji Pethraj Shah Medical College and Guru Govind Govt. hospital, Jamnagar for their advice and support. The authors are grateful for the cooperation of those patients who participated in this study

REFERENCES

1. Flint J, Harding RM, Boyce AJ, Clegg JB. The population genetics of the haemoglobinopathies. *Baillieres Clinical Haematol.* 1998;11(1):1-51.
2. Tong MJ, El-Farra NS, Reikes AR, Co RL. Clinical outcomes after transfusion-associated hepatitis C. *N Engl J Med.* 1995;332(22):1463-1466.
3. di Bisceglie AM, Goodman ZD, Ishak KG, Hoofnagle JH, et al. Long-term clinical and histopathological follow-up of chronic posttransfusion hepatitis. *Hepatology.* 1991;14(6):969-974.
4. Wong JB, McQuillan GM, McHutchison JG, Poynard T. Estimating future hepatitis C morbidity, mortality, and costs in the United States. *Am J Public Health.* 2000;90(10):1562.
5. Global surveillance and control of hepatitis C WH. Report of a WHO consultation organized in collaboration with the Viral Hepatitis Prevention Board, Antwerp, Belgium. *J Viral Hepat.* 1999;6(1):35-47.
6. Mansour AK, Aly RM, Abdelrazek SY, Elghannam DM, et al. Prevalence of HBV and HCV infection among multi-transfused Egyptian thalassemic patients. *Hematol Oncol Stem Cell Ther.* 2012;5(1):54-59.
7. Cappellini MD, Cohen A, Eleftheriou A, Piga A, et al. Guidelines for the clinical management of thalassaemia. 2008.
8. Borgna-Pignatti C, Galanello R. Thalassemias and related disorders: quantitative disorders of hemoglobin synthesis. *Wintrobe's Clinical Hematology.* 2004;42:1319-1365.

9. Hussain H, Iqbal R, Khan MH, Iftikhar B, et al. Prevalence of hepatitis C in beta thalassaemia major. *Gomal J Med Sci.* 2008;6(2).
10. Madan N, Sharma S, Sood SK, Colah R. Frequency of β -thalassemia trait and other hemoglobinopathies in northern and western India. *Indian J Hum Genet.* 2010;16(1):16.
11. Colah RB, Gorakshakar A. Control of thalassemia in India. *Thalassemia Reports.* 2014;4(2):1955.
12. Bhavsar H, Patel K, Vegad M, Madan M, et al. Prevalence of HIV, hepatitis B and hepatitis C infection in thalassemia major patients in tertiary care hospital, Gujarat. *Nat J Integr Res Med.* 2011;2(3):47-51.
13. Mukherjee K, Bhattacharjee D, Chakraborti G. Prevalence of hepatitis B and hepatitis C virus infection in repeatedly transfused thalassemics in a tertiary care hospital in eastern India. *Int J Res Med Sci.* 2017;5(10):4558-62.
14. Modi D, Rathod GB, Delwadia KN, Goswami HM. Study of seroprevalence in thalassemic patients. *Int Arch Integr Med.* 2016;3:57-65.
15. Surani CC, Shah RV, Sinha M. A study of prevalence of hepatitis-B and hepatitis-C infection in thalassemic patients in a tertiary care hospital, Jamnagar, Gujarat, India. *Int J Curr Microbiol App Sci.* 2018;7:3142-3146.
16. Agrawal S, Sulaniya PK, Garg K, Choudhary R, et al. Seroprevalence of hepatitis-c infection in multi-transfused thalassemic children: study from a West Indian tertiary care center. *Int J Contemp Pediatrics.* 2017;4(5):1871-1874.