

Incidence of Viral Hepatitis in Thalassaemic Patients As A Consequence Of Multiple Blood Transfusions

Gira P. Mankad¹ and S.P. Singh²

M.V.M.Science and Homescience college, Rajkot
Department of Biosciences, Saurashtra University, Rajkot
Email: gira_mankad@rediffmail.com

ABSTRACT: There are many blood borne transfusion transmitted diseases. The most common among them is undoubtedly viral hepatitis B and hepatitis C. Patients with the history of multiple blood transfusions are at much greater risk of infection by contaminated blood products. Thalassaemia major is one such condition where repeated blood transfusions are required. In our study a total of 218 patients were screened for Hepatitis B surface antigen and HCV antigen in their serum. 08 (3.66%) were found to be HBsAg positive and 18(8.25%) patients were found to be HCV positive by third generation ELISA technique. This was compared to the data of donor population which were considered healthy at the time of blood donation. HBsAg seropositivity was 1.98% and HCV seropositivity was 1.09% in this population. This result clearly indicates high incidence of transfusion transmitted hepatitis in thalassaemic patients and much higher incidence of HCV infection compare to donor population is a matter of concern and research.

I. INTRODUCTION

Thalassaemia describes a group of inherited disorders characterized by reduced or absent amounts of hemoglobin, the oxygen-carrying protein inside the red blood cells. Thalassemiias are classified according to the globin that is affected, hence the names *alpha* and *beta* thalassaemia. Beta thalassaemia may be the most well-known type of thalassaemia and is also called Cooley's anemia. Beta thalassaemia major usually causes severe anemia that can occur within months after birth. If left untreated, severe anemia can result in insufficient growth and development, as well as other common physical complications that can lead to a dramatically decreased life-expectancy. Fortunately, in developed countries beta thalassaemia is usually identified by screening in the newborn period, before symptoms have developed. Children who are identified early can be started on ongoing blood transfusion therapy as needed. Individuals with beta thalassaemia major receive regular blood transfusions, usually on a monthly basis. This helps prevent severe anemia and allows for more normal growth and development. Transfusion therapy does have limitations. Although blood supplies in most part of the world are very safe, particularly relative to the past there remains an increased risk of exposure to such blood-borne infections as hepatitis. Most deaths caused by blood transfusion worldwide are due to the transmission of infectious agents: viruses, bacteria or protozoa.

Evolving suburban wilderness and global travel ensure the emergence and spread of 'new' blood borne pathogens. Microbial adaptation, climate and weather changes, war and famine and the specter of bio-terrorism all raise the concern of emerging infection threats to the blood supply. The agents responsible share the following characteristics: persistence in donor's bloodstream, giving rise to carrier or latent states, a susceptible recipient population, the ability to cause asymptomatic infections, stability in stored blood and in many cases in plasma fractions. Ideally, blood for transfusion should either be tested for all pathogens that are prevalent in a given population and cause serious disease or treated to inactivate all such pathogens. In practice neither is possible. (Mollison et al 1997)

Hepatitis B (HBV) and hepatitis C virus (HCV) are transmissible by the parenteral route and may be found in blood and other body fluids. From the bloodstream, the viruses travel to the liver where they replicate in hepatocytes, resulting in an acute or chronic liver infection. (Dodd RY. 2007)

II. MATERIALS AND METHODS

A group of 121 patients suffering from beta thessemia at K.T. Children Hospital, Rajkot initially included in the study. At the end of the study we have registered 218 patients.

These patients had been receiving blood transfusions regularly at K.T. Children Hospital

from 2005 till the end of the study period. Patients who had received at least 2 previous blood transfusions were included for serological follow up for 5 successive years. Transfusion and clinical records of all patients were maintained. About 3 ml serum sample was collected before transfusion during a specific period (March-April-May) of each year and samples were preserved.

Serological study: Frozen samples were tested after the study period for various viral markers in the same laboratory by one person using the same batches of reagents and kits. Tests were carried out by commercially available, third generation, enzyme linked immunosorbent assay (ELISA) for the following TTD markers: (i) HBsAg (Microscreen HbsAg ELISA Test Kit by Span Diagnostics) (ii) antibodies to HCV (SP- NANBASE C-96 3.0 test kit by General Biological Corp.)

III. RESULTS AND DISCUSSION

Present study was conducted to observe incidence of viral infection in thalassemic patients who received multiple blood transfusion in and around Rajkot city. Rate of viral infections among thalassemic patients were compared with incidence of these infections in donor population. Table 1 shows prevalence of viral infections in thalassemic patients at the beginning of the study in 2005 the rate of infection was out of 121 only 04 were HBsAg Positive and 07 were HCV Positive while in 2010 out of 218 patients 8 were HBsAg positive and 18 were HCV Positive. Rate of HBsAg positivity among donor population is 1.71% in 2005 and 1.98% in 2010 while HCV seropositivity was 0.87% at the beginning of the study and 1.09% at the end the study (Figure 1). Rate of HBsAg seropositivity in thalassemic patients was 3.3% initially and 3.66% at the end of the study while it was 5.78% in 2005 and 8.25% in 2010 for HCV marker. (Figure2).

Rate of HBsAg seropositivity among blood donors vary from 0.40% (Graves and Biswas, 1973) to 17.70% (Talib, 1983). It was found to be varying from 2.0% (Chakravarti, 2005) to 13.8% (Mollah A.H., 2003) in patients suffering from thalassemia.

Rate of Hepatitis C infection was found to be varying from 0.74% (DeSilva, 1998) to 2.4% (Khan M., 1993) in blood donors while it was between 5.1% (Samimi-Rad K. 2007) to 19.3% (Mirmomen S., 2006) in thalassemic patients which is comparable to our data.

Hepatitis B virus infection is a major cause of morbidity and mortality in humans and it is endemic all over the world. (Blumberg, 1965) About 350 million people of the world are infected with this virus (Lee, 1997). Hepatitis B virus is major cause of chronic hepatitis, cirrhosis and hepatocellular carcinoma especially in Asian countries. (Tabore et al 1980)

Hepatitis C virus first identified in 1989, was major cause of non-A, non-B hepatitis. Hepatitis C Virus has been identified as an important etiological agent responsible for transfusion associated hepatitis and accounts for about fifty percent of the sporadic cases of non-A, non-B hepatitis (CDC, 1991).

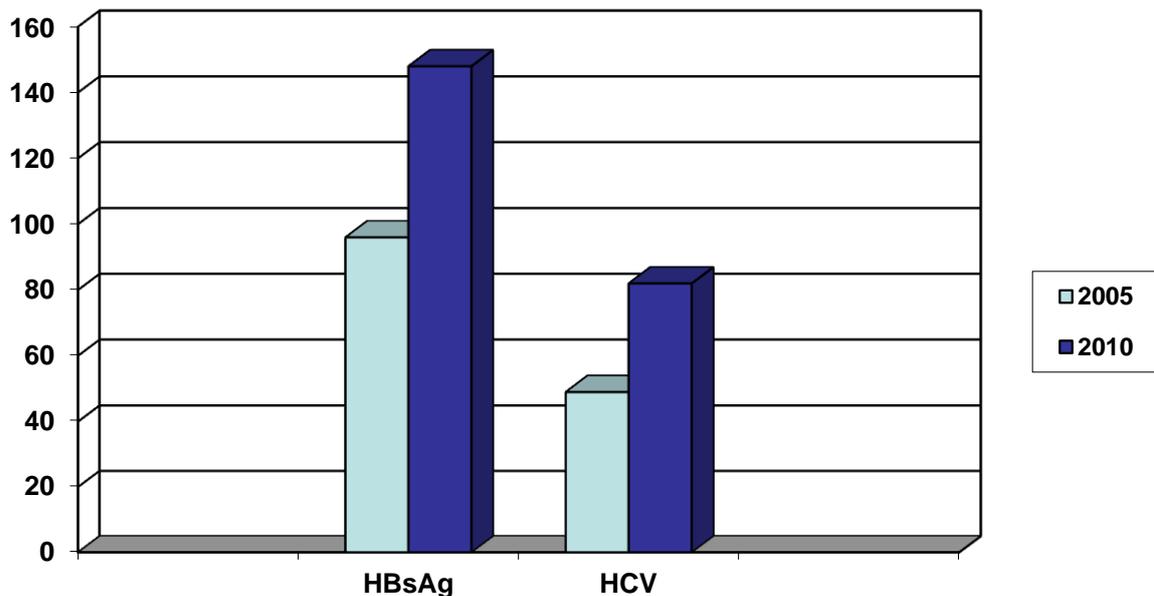
Around 100 million people worldwide are estimated to be infected with HCV. Chronicity occurs in about 80% of infected patients. The health burden of chronic hepatitis C infection in the western world is gradually being realized. In most Asian countries, Hepatitis B virus is still the major cause of chronic liver disease and hepatocellular carcinoma. In Japan, the pattern is changing in the past decades and now HCV is the predominant cause of HCC. A comprehensive assessment of HCV infection in Asia is important. Any factor or activity that increases the risk of transmission must be identified and built into strategy for infection control. This is particularly vital when effective HCV vaccine is not available. (Serin and Kunio 2002).

Risk of infectious complications due to blood transfusion is a major concern. Number and types of microbial infections depend on variety of factors as discussed. In developing countries like India where medical facilities are sporadic and not up to the best of the standards such incidences are obviously high. The risk is increased manifold if a person receives multiple blood transfusions.

References

- [1] Alter H. J. (1988.) Transfusion associated non-A, non-B hepatitis: the first decade.; In *Viral hepatitis and liver disease*. (Ed: Zuckerman A.J.), Alan R. Liss, Inc., New York,
- [2] Blumberg B.S., Alter H.J. and Visnich S.A., (1965) "New" Antigen in Leukemia sera, *JAMA*. 191, (7), 541-546.
- [3] CDC (1991) Hepatitis B virus – A comprehensive strategy for eliminating transmission in the U.S. through universal vaccination. *MMWR* 40(RR 13): 1-19.
- [4] Chakravarti A., Verma V., Kumaria R., et. al., **Anti-HCV seropositivity among multiple transfused patients with beta thalassaemia**. *J. Indian Med. Assoc.* 2005; 103(2) : 64-66.
- [5] DeSilva H.J., Foreska M.M.D., Zoysa N.S. et. al., **Seroepidemiology of Hepatitis B and C in Sri Lanka in: Transfusion Associated Hepatitis**. (Ed: Hess G) New Delhi, CBS publication; 1998: 116-25.
- [6] Dodd RY.: Current risk for transfusion-transmitted infections. *Current Opinion in*
- [7] *Hematology*, 2007, 14(6):671–676.
- [8] Grave I.L and Biswas S.K., The frequency and persistence of hepatitis associated antigen in Calcutta blood donors. *Ind. J. Med. Res.* 1974; 384.
- [9] Khan M., Hussain M., Yano M. et. al., Comparison of seroepidemiology in blood donors between Bangladesh and Japan. *Gastroenterol. Jpn.* 1993; 28(5): 28-31.
- [10] Lee W., Hepatitis B virus infection. *N Engl. J. Med.* (1997) 337: 1733-1745.
- [11] Mirmomen S., Alavian S.M., Hajarizadeh B., et. al., Epidemiology of hepatitis B, hepatitis C, and human immunodeficiency virus infections in patients with beta-thalassemia in Iran: a multicenter study. *Arch Iran Med.* 2006; 9(4) : 319-323.
- [12] Mollah A.H., Nahar N., Siddique M.A. et. al., **Common transfusion-transmitted infectious agents among thalassaemic children in Bangladesh**. *J. Health Popul. Nutr.* 2003; 21(1): 67-71.
- [13] Mollison P.L., Engelfriet, C.P., Contreras, M. et. al. (1997), In: *Blood Transfusion in Clinical Medicine*, 10th Ed. (Eds: Mollison P.L., Engelfriet, C.P., Contreras, M. et al.) Blackwell Science, London: 116-132.
- [14] Sarin S.K., Kunio O., **Hepatitis B and C Carrier to Cancer (2002)**. (Ed: Sarin S.K) Harcourt (India) Pvt. Ltd. New Delhi. 9
- [15] Tabor E. (1982) **Infectious Complications of Blood Transfusion**. (Ed: Tabore E.): Academic Press New York, 134-170.
- [16] Talib V.H., Halder S.N., Khurana S.R. et. al., Hepatitis B surface antigen in blood donors, general population and in cases of infective hepatitis. *J. Clinician.* 1983; 47: 89.

Graph 1



Donor's Status

Table 1

Year	Total	HBsAg +Ve	%	HCV +Ve	%
2005 (at the beginning of Study	5586	96	1.71%	49	0.87%
2010 (at the end of Study)	7458	148	1.98%	82	1.09%

Graph 2

Prevalence of viral infections in Thalassemic patients

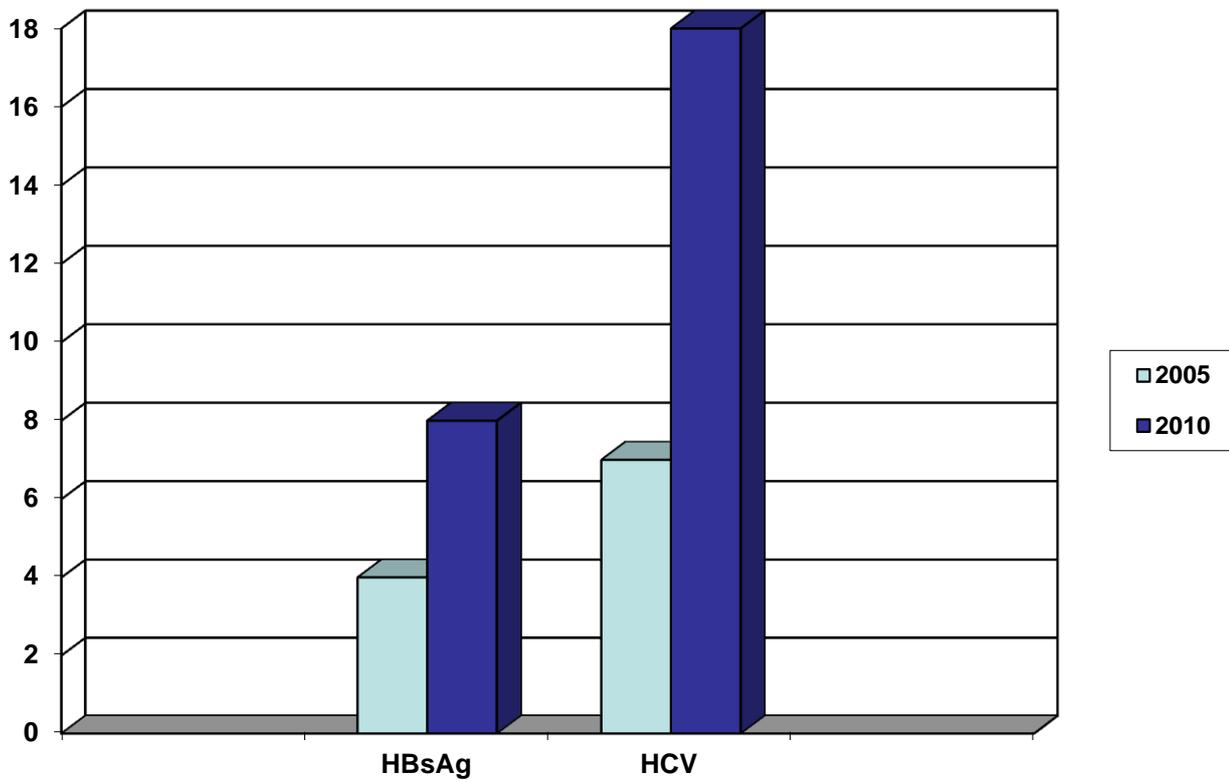


Table 2

Year	Total	HBsAg +Ve	%	HCV +Ve	%
2005 (at the beginning of Study	121	04	3.3%	07	5.78%
2010 (at the end of Study)	218	08	3.66%	18	8.25%