

## Reverse Vertical Transmission of Hepatitis-B from Transfusion-infected Children to Biological Mothers

**RAJEEV KHANNA,\*EKTA GUPTA AND SEEMA ALAM**

*From Departments of Pediatric Hepatology and \*Virology, Institute of Liver and Biliary Sciences, Vasant Kunj, New Delhi, India.*

*Correspondence to:*

*Dr Rajeev Khanna, Assistant Professor,  
Department of Pediatric Hepatology,  
Institute of Liver and Biliary Sciences,  
D-1, Vasant Kunj, New Delhi 110 070, India.*

*drrajeev\_khanna@rediffmail.com*

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**Background:** Perinatal and horizontal are the common modes of transmission of hepatitis-B virus in children. **Case characteristics:** Two mother-child pairs with children having received multiple blood transfusions in past. **Observation:** Both the mothers developed acute hepatitis-B infection whereas children were demonstrated to be having chronic infection with hepatitis-B. **Outcome:** One mother cleared her hepatitis-B infection whereas it persisted in the other. Both children required anti-viral treatment. **Message:** Hepatitis-B virus may rarely get transmitted from infected children to their mothers causing acute infection.

**Keywords:** *Hepatitis, Perinatal Transmission, Vertical transmission.*

**V**ertical or mother-to-child transmission (MTCT) is the predominant mode of transmission (>50%) of hepatitis-B virus (HBV) in areas of moderate and high endemicity [1,2]. Horizontal transmission of HBV, through infected blood products or contaminated syringes, is seen in 24-80% of cases in Asian countries [1,3]. Reverse vertical transmission of HBV is a rare phenomenon [4]. Here, we report two mother-child pairs where HBV possibly got transmitted from children to their biological mothers.

**Case 1:** This boy was born at full term to an HBsAg negative mother (unimmunized against HBV) through cesarean section with uneventful perinatal course. He received 3 doses of HBV vaccine at 6, 10 and 14 weeks, and was exclusively breastfed till 6 months. There were two previous pregnancies – first was a miscarriage and second was a live female, who was HBsAg negative at 3.5 years of age. Child remained well till 11 months of life, when he developed acute urinary retention, and was diagnosed as having rhabdomyosarcoma of urinary bladder. He received 49 weeks of chemotherapy along with 28 sessions of radiotherapy and 5 packed red cell transfusions. After 8 months of his last transfusion, mother developed acute hepatitis B infection. Her infection resolved uneventfully within a span of four weeks. She had history of ear-piercing, but no prior history of jaundice, blood transfusion, tattooing, dental procedure, surgery or multiple unprotected sexual contacts. Father and other close contacts were negative for HBsAg. Child was then detected to have chronic

hepatitis-B in immunoclearance phase (**Table I**). He is currently on sequential treatment with lamivudine and interferon.

**Case 2:** This male child was born to an HBsAg negative primipara at 28 weeks gestation. He was first of twin delivered vaginally; the second twin was a stillborn delivered through cesarean section. Antenatally, mother had preeclampsia in the late second trimester, and had fever with premature rupture of membranes 24 hours prior to delivery. Neonate was admitted in neonatal intensive care unit for 20 days where he was managed for respiratory distress syndrome, early onset sepsis, necrotizing enterocolitis and hypothermia, and was given 7 fresh frozen plasma transfusions for coagulopathy. Subsequently, he received immunization against HBV at 6, 10 and 14 weeks. Breastfeeding was continued for 7 months. Seven months after delivery, mother developed acute hepatitis-B infection (**Table I**). Her laboratory parameters normalized within 6 weeks, followed by development of protective antibodies after 6 months (anti-HBs 67 mIU/mL). She had no prior history related to high risk behavior, and was unvaccinated against HBV. Child was subsequently diagnosed to have chronic hepatitis-B in immuno-clearance phase. All other family members were HBsAg negative. Child was started on standard interferon alpha-2a for 24 weeks. He attained seroconversion at the end of therapy (**Table I**).

### DISCUSSION

Horizontal transmission from adopted children infected with HBV to their family members has been reported

**TABLE I** LABORATORY PARAMETERS IN TWO MOTHER-CHILD PAIRS

	<i>Mother-child pair 1</i>		<i>Mother-child pair 2</i>	
	<i>Child 1</i>		<i>Child 2</i>	
	<i>At detection</i>	<i>Follow-up</i>	<i>At detection</i>	<i>Follow-up</i>
Age (mo)	24	29	19	31
Bilirubin (mg/dL)	0.3	0.5	0.46	0.5
ALT (IU/L)	58	29	289	28
IgM Anti-HBc	Negative	–	Negative	–
HBeAg	Positive	–	Positive	Negative
Anti-HBe	Negative	–	Negative	Positive
HBV DNA levels (IU/mL)	$1.1 \times 10^8$	$1.85 \times 10^6$	$3.25 \times 10^5$	$5.47 \times 10^2$

  

	<i>Mother 1</i>		<i>Mother 2</i>	
	<i>During acute hepatitis</i>	<i>Follow-up at 3 mo</i>	<i>During acute hepatitis</i>	<i>Follow-up at 12 mo</i>
	HBsAg	Positive	Positive	Positive
Bilirubin (mg/dL)	10.3	1.3	17.5	0.33
ALT (IU/L)	2189	18	5062	15
IgM Anti-HBc	Positive	–	Positive	–
HBeAg	Positive	–	Positive	Negative
Anti-HBe	Positive	–	Negative	Positive
HBV DNA levels (IU/mL)	$9.31 \times 10^5$	$6.5 \times 10^1$	–	Not detected

ALT = Alanine aminotransferase; Anti-HBe = Antibody against HBeAg; IgM Anti-HBc = IgM antibody against core antigen.

earlier [5,6]. This is explainable by the demonstration of HBV in tears, saliva, sweat and urine of children, and the possible modes of transmission are close household skin-to-skin contact, breastfeeding, sharing of toothbrushes, and exposure to open wound and blood spills [7]. However, reverse vertical transmission – from child to biological mother during postnatal period or late in childhood – is a rare phenomenon. There is an earlier report [4] where mother of one of the three HBV-infected newborns got the virus and developed acute hepatitis-B. In the present report, we described two such scenarios, where the transfusion-infected children possibly transmitted HBV to their non-immunized mothers, and both women developed acute hepatitis-B. We hypothesize the possible mode of transmission in both of our cases to be close household contact and additionally – in the second case – breastfeeding by the infant. However, unlike the previously reported case, full length nucleotide sequencing was not done in our cases which could have excluded a rare chance of laboratory cross-contamination [4].

As both of our children received HBsAg – tested blood products from certified blood banks, the donors might either be in the window phase of infection or had occult HBV infection. A recent study reported

transmission of HBV in 28% of cases due to occult HBV infection. Presence of donor Anti-HBs reduced risk by 5-fold, whereas transfusion of fresh frozen plasma, in comparison to packed red cells, increased risk by 9-fold [8]. Recommendations are available to interrupt the transfusion from blood products in this manner [8]. We could not look for anti-HBc, anti-HBs titre or HBV-DNA in the transfused blood products, but with negative maternal HBsAg status during pregnancy and absence of any household exposure, we assumed that both the children got infected by the transfused blood products. Also, we did not know the anti-HBs and anti-HBc status of the first child before initiation of chemotherapy, which could have better indicated his protection level and risk of HBV reactivation after immunosuppression [9].

In India, there is still a large population of non-immune children as well as mothers who remain susceptible to the virus [10]. The present case report brings emphasis on the screening and timely vaccination of all the members of the family of infected children.

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## REFERENCES

1. Sokal EM, Paganelli M, Wirth S, Socha P, Vajro P, Lacaille F, *et al.* Management of chronic hepatitis B in childhood: ESPGHAN clinical practice guidelines: Consensus of an expert panel on behalf of the European Society of Pediatric Gastroenterology, Hepatology and Nutrition. *J Hepatol.* 2013;59:814-29.
2. WHO. Position Paper on Hepatitis B, 2009. Available From: [www.who.int/wer/2009/](http://www.who.int/wer/2009/). Accessed February 1, 2014.
3. Lok ASF, McMahon BJ. AASLD Practice Guidelines. Chronic Hepatitis B – Update 2009. *Hepatology.* 2009;50:1-36.
4. Niederhauser C, Candotti D, Weingand T, Maier A, Tinguely C, Stolz M, *et al.* Reverse vertical transmission of hepatitis B virus (HBV) infection from a transfusion-infected newborn to her mother. *J Hepatol.* 2012;56:734-7.
5. Sciveres M, Maggiore G. Hepatitis B “by proxy”: An emerging presentation of chronic hepatitis B in children. *J Pediatr Gastroenterol Nutr.* 2007;44:268-9.
6. Sokal EM, Collie OV, Butts JP. Horizontal transmission of hepatitis B from children to adoptive parents. *Arch Dis Child.* 1995;72:191.
7. Komatsu H, Inui A, Sogo T, Tateno A, Shimokawa R, Fujisawa T. Tears from children with chronic hepatitis B virus (HBV) infection are infectious vehicles of HBV transmission: Experimental transmission of HBV by tears, using mice with chimeric human livers. *J Infect Dis.* 2012;206:478-85.
8. Allain JP, Mihaljevic I, Gonzalez-Fraile MI, Gubbe K, Holm-Harritshøj L, Garcia JM, *et al.* Infectivity of blood products from donors with occult hepatitis B virus infection. *Transfusion.* 2013;53:1405-15.
9. Shouval D, Shibolet O. Immunosuppression and HBV reactivation. *Semin Liver Dis.* 2013;33:167-77.
10. Verma R, Khanna P, Prinja S, Rajput M, Chawla S, Bairwa M. Hepatitis B Vaccine in national immunization schedule: A preventive step in India. *Hum Vaccine.* 2011;7:1387-8.

## Retroaortic Left Renal Vein with Cascade of Complications in a Neonate

SANDESH GULERIA, JYOTI SHARMA AND SANJEEV CHAUDHARY

From the Department of Pediatrics, Dr. RPGMC, Tanda, Kangra (HP), India.

Correspondence to:

Dr. Sanjeev Chaudhary, Professor,  
Department of Pediatrics,  
Dr. RPGMC, Tanda, Kangra (HP), India.

[s\\_chaudhary@ymail.com](mailto:s_chaudhary@ymail.com)

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**Background:** Retroaortic left renal vein, is a rare congenital anomaly. **Case characteristics:** A 14-day-old male neonate with retroaortic left renal vein with posterior nutcracker phenomenon resulting in renal congestion. **Observation:** He developed septicemia, renal abscess and thrombosis of abdominal aorta. **Outcome:** Improvement on antibiotics and heparin. **Message:** Retroaortic left renal vein can cause life threatening complications.

**Keywords:** Neonate, Renal vein, Thrombosis.

**A** retroaortic left renal vein (RLRV) is located between the aorta and lumbar vertebrae, and drains into the inferior vena cava (IVC) or left common iliac vein [1]. Compression of the left renal vein between the abdominal aorta and vertebrae leads to haematuria, flank pain, varicocele and abdominal pain; this is also called posterior nutcracker phenomenon [2]. Congested kidney and renal infarcts secondary to posterior nutcracker phenomenon may lead to bacterial localization and abscess formation. Aortic thrombosis is a recognized complication of infection and sepsis [3]. Computed tomography (CT), magnetic resonance imaging and ultrasonography (USG) are effective for detection of this congenital anomaly [1]. We present a neonate with RLRV with posterior nutcracker phenomenon who subsequently developed sepsis and thrombosis of abdominal aorta.

### CASE REPORT

A 14-day-old male neonate was admitted with history of lump abdomen and excessive cry for 4 days and progressively enlarging lump in left side of abdomen for 2 days. There was no history of fever, lethargy, poor feeding, vomiting, seizure or any respiratory symptom. There was no bowel or urinary complaints. Perinatal period was uneventful.

The infant was irritable with normal general physical examination and stable vitals. There was a hard, non-mobile, 4×3 cm lump present in left hypochondrium and lumbar regions. Rest of the systemic examination was normal.

Investigations revealed neutrophilia and deranged renal function (urea 177 mg/dL; creatinine 1.4 mg/dL).