ian hyperstimulation syndrome. N Engl J Med. 2003;349(8):753-9.
7. Smits G, Olatunbosun O, Delbaere A, Pierson R, Vassart G, Costagliola S. Ovarian hyperstimulation syndrome due to a mutation in the follicle-stimulating hormone receptor. N Engl J Med. 2003;349(8):760-6.
8. Grumbach MM, Styne DM. Puberty: ontogeny, neuroendocrinology, physiology, and disorders. In: Wilson JD, Foster DW, Kronenberg HM. et al., editors. Williams Textbook of Endocrinology. Philadelphia: Saunders;1998. pp. 1593-594.
9. Barnes ND, Hayles AB, Ryan RJ. Sexual maturation in juvenile hypothyroidism. Mayo Clin Proc. 1973;48(12):849-56.
10. Aboulghar M. Prediction of ovarian hyperstimulation syndrome (OHSS). Estradiol level has an important role in the prediction of OHSS. Hum Reprod 2003;18(6):1140-1.
11. Shoham Z, Balen A, Patel A, Jacobs HS. Results of ovulation induction using human menopausal gonadotropin or purified follicle-stimulating hormone in hypogonadotropic hypogonadism patients. Fertil Steril 1991;56(6):1048-53.
12. Shimon I, Rubinek T, Bar-Hava I, Nass D, Hadani M, Amsterdam A, Harel G. Ovarian hyperstimulation without elevated serum estradiol associated with pure follicle-stimulating hormone-secreting pituitary adenoma. J Clin Endocrinol Metab 2001;86(8):3635-40.

Prevalence of GBV-C/ hepatitis G virus viremia among chronic hepatitis B, chronic hepatitis C and hemodialysis patients in Turkey

To the Editor: The newly discovered hepatitis G virus (HGV) or GBV-C are isolates of the same virus, which is a single-stranded RNA virus of positive polarity with 9362 nucleotides.[1] It can be transmitted via blood transfusion and intravenous drug use, sexually, and from an infected mother to her child.[2] High prevalences of GBV-C/HGV have been found in subjects with frequent parenteral exposure and in groups at high risk of exposure to blood and blood products, including drug abusers, hemodialysis patients, multitransfused individuals and haemophiliacs.[3] Due to shared risk factors, coinfection of GBV-C/HGV with hepatitis B (HBV) or hepatitis C (HCV) viruses in chronically infected patients has been reported at frequencies ranging from 10 to 25%.[4]

Because the prevalence of GBV-C/HGV is unclear in Turkish population, we sought analyze the prevalence of GBV-C/HGV-RNA in the sera of different groups in the Turkish population. Three hundred and ten Turkish serum samples classified into four groups were studied. Sera of 85 hemodialysis patients, 80 chronic hepatitis B patients, 75 chronic hepatitis C patients, and 70 healthy persons (control group) were tested for the presence of GBV-C/HGV-RNA. The control group included apparently healthy individuals who had participated in occupational screening for a randomly selected viral hepatitis marker. Thirty-seven were male and 33 female with a mean age of 42.5±11.8 years (range, 20-65 years). None were positive for anti-HCV or for HBsAg.

RNA was extracted from 150 µl of serum using the Nucleospin Virus Kit (Biogene, Kimbolton, UK). Real-time PCR was performed using primer pairs and a probe located in the 5′ untranslated region (5′UTR) of GBV-C/HGV-RNA using the ABI Prism 7700 Sequence Detector System (Perkin Elmer, Foster City, Calif.). Data were analysed by Fisher' s exact test. A *P* value less than 0.05 was considered significant.

GBV-C/HGV-RNA was detected in 52 of the 310 sera tested with an overall prevalence of 17%. The highest prevalence was encountered among chronic hepatitis B patients (28%) followed by hemodialysis patients (24%), chronic hepatitis C patients (6%), whereas the lowest prevalence rate of 4% was detected among healthy persons (Table 1). HGV was significantly more frequent in chronic hepatitis B patients, hemodialysis patients, and chronic hepatitis C patients than in healthy persons ($P<0.05$).

It has been documented that patients with chronic hepatitis often harbor more than one hepatitis agent.[5] The apparent link between hepatotropic viral infections probably reflects common exposure and transmission patterns rather than a specific interdependence relation. Heringlake et al.[6] reported a striking high prevalence of HGV-RNA among patients with viral hepatitis B, C and D reaching 16%, 20% and 36%, respectively. In our study, the prevalence rate of GBV-C/HGV was 7% in the chronic hepatitis C group. The relative low prevalence of GBV-C/HGV-RNA in this group may be explained by a similar reciprocal replication pattern among patients coinfected with HBV and HCV.[7] This had been proposed by Raimondo et al.,[8] who suggested that while long-lasting persistance of HCV is the rule in chronically infected individuals, clearence of GBV-C/HGV after years of chronic infection is a frequent event.

Table 1. GBV-C/HGV RNA positivity among the four groups.

Group	No. tested	HGV RNA	
		No. positive	%
Chronic hepatitis B patients	80	23	29
Chronic hepatitis C patients	75	5	7
Hemodialysis patients	85	21	25
Healthy controls*	70	3	4
Total	**310**	**52**	**17**

*$P<0.05$ versus other groups.

Several authors demonstrated a high prevalence of HGV in hemodialysis patients with a wide range between different countries.[9] Our figure of 25% is similar to that observed in most countries, including Italy[10] and Spain.[11] However, lower and higher prevalences have been reported in other countries: 3% to 8% in Japan and Germany[12,13] and 55% to 57% in Indonesia and France.[14,15] These differences in the prevalence of GBV-C/HGV may be explained by epidemiological variations, including a variable rate of blood transfusions and a variable adherence to universal precautions. However, a methodological reason may also contribute to this variability. In the reported studies, most determinations are performed using noncommercial tests and different primers were amplified. An available commercial kit or at least unified criterion for the detection should be necessary to obtain useful data to compare the prevalence of GBV-C/HGV.

In conclusion, GBV-C/HGV-RNA is highly prevalent among the different Turkish patient populations, being highest in chronic hepatitis B patients. Although much information has been learned about GBV-C/HGV infection in the short time since the discovery of the virus, the clinical and pathological significance of this infection needs better evaluation, particularly in patients infected with other hepatitis viruses, and in hemodialysis patients.

Sinem Akcali
Tamer Sanlidag
Beril Ozbakkaloglu

Correspondence:
Sinem Akcali, MD
Celal Bayar University
Microbiology and Clinical
Microbiology
Koruturk mah. Lodos sok. No:9
D:3
Balcova Izmir 35330 Turkey
akcalisinem@yahoo.com

References

1. Kar P, Bedi P, Berry N, et al. Hepatitis G virus (HGV) infection in voluntary and commercial blood donors in India. Diagn Microbiol Infect Dis. 2000; 38: 7-10.
2. Fabrizi F, Lunghi G, Bacchini G, et al. Hepatitis G virus infection in chronic dialysis patients and kidney transplant recipients. Nephrol Dial Transplant. 1997; 12: 1645-51.
3. Sathar MA, Soni PN, Pegoraro R et al. A new variant of GB virus C/hepatitis G virus (GBV-C/HGV) from South Africa. Virus Research. 1999; 64: 151-60.
4. Bassit L, Bernhard K, Ribeiro-dos-Santos G et al. Hepatitis G virus: Prevalence and sequence analysis in blood donors of Sao Paulo, Brazil. Vox Sang. 1997; 74: 83-7.
5. Linnen J, Wages J Jr, Zhang-Keck ZY et al. Molecular cloning and disease association of hepatitis G virus: a transfusion-transmissible agent. Science.1996; 26: 505-8.
6. Heringlake S, Tillmann HL, Cordes-Temme P et al. GBV-C/HGV is not the major cause of autoimmune hepatitis. J Hepatol. 1996; 25: 980-4.
7. El-Zayadi AR, Abe K, Selim O et al. Prevalence of GBV-C/hepatitis G virus viraemia among blood donors, health care personel, chronic non-B non-C hepatitis, chronic hepatitis C and hemodialysis patients in Egypt. J Virol Methods. 1999; 80: 53-8.
8. Raimondo G, Pernice M, Longo G. Hepatitis G virus in liver disease: Cause or case? Ital J Gastroenterol. 1996; 28: 470-3.
9. Beccari M, Sorgato G. The new HGV/HGVB-C virus in dialysis patients. J Nephrol. 1998; 11: 165-8.
10. Sampietro M, Badalamenti S, Graziani G et al. Hepatitis G virus infection in hemodialysis patients. Kidney Int. 1997; 51: 348-52.
11. Forns X, Fernandez-Llama P, Costa J et al. Hepatitis G virus infection in a hemodialysis unit: prevelance and clinical implications. Nephrol Dial Transplant. 1997; 12: 956-60.
12. Masuko K, Mitsui T, Iwano K et al. Infection with hepatitis GB virus C in patients on maintenance hemodialysis. N Engl J Med. 1996; 334: 1485-90.
13. Ideura T, Tanaka E, Nakatsuji Y et al. Clinical significance of hepatitis G virus infection in patients on long-term hemodialysis. J Gastroenterol Hepatol. 1997; 12: 762-5.
14. Tsuda F, Hadiwandowo S, Sawada N et al. Infection with GB virus C (GBV-C) in patients with chronic liver disease or on maintenance hemodialysis in Indonesia. J Med Virol. 1996; 49: 248-52.
15. de Lamballerie X, Charrel RN, Dussol B. Hepatitis GB virus C in patients on hemodialysis. N Engl J Med. 1996; 334: 1549.

Surfactant protein B deficiency: a rare cause of respiratory failure in a Lebanese newborn

To the Editor: Respiratory diseases secondary to congenital surfactant proteins deficiency are increasingly recognized. To bring to the attention of pediatricians an unusual cause of neonatal respiratory disease, we report on a newborn with progressive respiratory disease due to surfactant protein B (SP-B) deficiency. To our knowledge, this is the first case of SP-B deficiency reported in the Middle East.

The patient was a term female newborn delivered vaginally after an uneventful pregnancy to second-degree consanguineous parents. The mother had a stillbirth and a newborn that died on the second day of life of respiratory causes. Four other siblings are normal. Apgar scores were 5 and 8 at 1 and 5 minutes, respectively. The baby was hypotonic and required vigorous stimulation. Birth weight was 3250 grams. Physical examination was remarkable for tachypnea, cyanosis and bilateral decreased air entry. Chest X-ray showed bilateral fine granular infiltrates.

The baby was started on antibiotics after a sepsis work up. She required conventional and then high frequency oscillatory ventilation because of hypoxemia and CO_2 retention. Echocardiography showed mild right ventricular hypertrophy. On the fourth day of life, she received bovine surfactant (Survanta, Abbott Laboratories, Columbus, Ohio, USA) intratracheally with clinical and radiological improvement that was not sustained on four additional doses. She then received furosemide, hydrocortisone and inhaled nitric oxide with no response. On the seventeenth day of life, she died of persistent hypoxemia with severe respiratory acidosis. Tracheal effluent collected before surfactant administration revealed complete absence of SP-B (Courtesy of