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.

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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

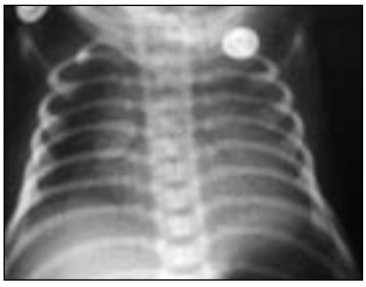

Figure 1. Chest x-ray showing bilateral fine granular infiltrates.

Dr. Jeffrey Whitsett, Cincinnati Children's Hospital). DNA analysis revealed the homozygous 122delT mutation, while both parents were heterozygous for the same mutation (Courtesy of Dr. Lawrence Nogee, Johns Hopkins University).

SP-B is a hydrophobic protein involved in the adsorption of surfactant phospholipids to the air-liquid interface. It is coded by a gene of 11 exons on chromosome 2. In 1993, Nogee et al reported SP-B deficiency causing severe respiratory disease, as described in our patient.[1] The patient and a sibling who had died earlier had a frame-shift mutation caused by a 2 base-pair insertion (121ins2) in exon 4 of the SP-B gene.[2] Radiologically, SP-B deficiency presents like hyaline membrane disease. Histopathologically, the distal airspaces appear filled with lipid-rich, periodic acid Schiff-positive, eosinophilic proteinaceous material.[1]

The diagnosis is established by failing to identify SP-B in the tracheal effluent and is confirmed by genetic studies, which show a mutation on the SP-B gene. More than 13 mutations have been described,[3] of which (121ins2) accounts for about 70%. Its frequency in the United States is estimated to be 1 per 1000-3000 individuals.[4] The 1043ins3 mutation was detected in 2 unrelated Pakistani families.[3] The mutation described in the present report (122delT) was described in a consanguineous kindred of Kurdish descent,[5] and in three unrelated Lebanese families (L. Nogee, personal communication). The recognition of specific mutations in various ethnic groups may allow diagnosis in individual patients and population-wide studies for the determination of gene frequency. This would gain particular importance in our population, where consanguinity is prevalent. SP-B deficiency is usually fatal, unless treated with lung transplantation.[6] Gene transfer therapy may be the treatment modality of the future.

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Serum immunoglobulin A, G and M in healthy adults in Dhofar, Oman

To the Editor: There is little data available on normal levels of serum immunoglobulin in the healthy adult populations of the Gulf countries and the Arab world. In many instances, the normal ranges for immunoglobulin, which are used by many hospitals within the Arab world, are those that are supplied by the manufacturer of the equipment or the reagents, and these values may not reflect the normal values of the local populations. Therefore, it is essential that each population establish its own normal values that can be used locally.

Although Oman has a climate that is generally hot and dry, similar to other Gulf Countries, Dhofar's (the southern region of Oman) climate is relatively cool and rainy, particularly during the summer monsoon. Individuals from this part of Oman may have their own distinct levels of immunoglobulin as this region has a distinct pattern of infections.[1]

Serum samples were collected from 489 (389 males and 100 females) Omani healthy adults from Dhofar recruited from healthy blood bank donors attending Sultan Qaboos Hospital in Salalah. Individuals with a history of acute or chronic illness, present or past allergy, parasitic infestation, chronic drug use, or present immunization were excluded from the study. After informed consent was secured, blood samples were obtained and allowed to clot at room temperature. Sera were separated and stored at -20°C until assayed for immunoglobulin G, M, and A, using a rate nephelometry system (Beckman Image System).