

make this conclusion. Similarly comparison of catch up of AGA and SGA infant was not possible.

The mean difference and their confidence interval suggest that developmental indices of VLBW infants were significantly lower than those of NBW infants.

Early age of assessment is the limitation of present study, and a much longer follow up might have been more informative. We would like to clarify that DQ of all VLBW infants was not above 90, it was the mean DQ of

this group. A mean DQ below 85 was observed in 22% of infants. This finding along with difference of 6 point in mean DQ between two groups cannot be underestimated and warrants a long-term follow up of these infant for their later outcomes. Also amongst babies who have a DQ above 90, it needs to be investigated, how these infants behave cognitively who have DQ of 90 as compared to those with 98.

SATISH SALUJA AND MANOJ MODI  
ssaluja@vsnl.com

## Vitamin D: The Emerging Superstar

There are certain issues that need to be emphasized in the recent review article on Vitamin D deficiency [1].

The authors' recommendation of 400 IU daily to toddlers and adolescents is erroneous. The current recommendation for this group is at least 600 IU per day [2]. Commercial preparations of 1000 IU per drop have the potential for Vitamin D toxicity.

The authors also state that "Supplementation in newborn period: For infants who are exclusively breastfed a minimum daily intake of 400 IU/day should be initiated within a few days after birth. Since most of the infant formulas contain 400 IU/L, infants who are on formula feeds also need supplementation unless they consume more than 1000 mL of formula per day."

Careful scrutiny of the commercial infant formulae available in the Indian market tells us a different story. Virtually no preparation has the concentration mentioned by the authors.

M SANKLECHA AND S SUNDARESAN  
Bombay Hospital Institute of Medical Sciences,  
Mumbai, India.  
doctormukesh@gmail.com

### REFERENCES

1. Balasubramanian S, Dhanalakshmi K, Amperayani S. Vitamin D deficiency in childhood – A review of current guidelines on diagnosis and management. *Indian Pediatr.* 2013;50:669-75.
2. Shah B, Finberg L. Single-day therapy for nutritional vitamin D-deficiency rickets: a preferred method. *J Pediatr.* 1994;125:487-90.

### REPLY

We agree that the recommended intake above 1 year is 600 IU as per Endocrine society guidelines. This has been taken into consideration in the article wherein the maintenance dose has been recommended as 600 to 1000 IU for 1 to 18 years old. Concentrated drops are best avoided as daily supplements because of risk of toxicity due to erroneous administration. Indian infant milk formulas provide vitamin D ranging from 288 to 378 IU/L, lower than the products available abroad.

S BALASUBRAMANIAN  
sbsped@gmail.com

## Influenza -B Associated Rhabdomyolysis With Acute Renal Failure

I read with interest the recent article "Influenza -B Associated Rhabdomyolysis with Acute Renal Failure" [1]. The boy developed dark urine with oliguria on day 5<sup>th</sup> of admission in consequent to right upper pneumonitis

caused by Influenza-B virus. The dark urine with renal failure could be due to hemoglobinuria or myoglobinuria. The authors have assumed it to be due to rhabdomyolysis leading to myoglobinuria based on striking elevation of serum creatine kinase (CK), LDH, AST/ALT only. Screening of urine should be done by Dipstick or Orthotoluidine blue test which will be indicative of hemoglobinuria, myoglobinuria, or hematuria. Absence of RBC on urine microscopic examination will rule out hematuria. Further urine

analysis should be done by electrophoresis or spectrophotometry to distinguish between myoglobinuria and hemoglobinuria. It is not clear from this case report whether blood examination for hemoglobin, Reticulocyte count, Direct Coomb's test were performed to rule out hemoglobinuria.

Myoglobinuria can lead to acute renal failure (ARF) by direct renal tubular damage or mechanical blockage of tubular lumen [2]. Hemoglobinuria also can behave in a similar way. After the development of ARF patient's serum creatinine level was 1.47 mg/dL. Serum urea nitrogen (BUN) was not studied at that time though both were normal soon after admission. BUN and serum creatinine ratio was important to know the type of ARF *i.e.* intrinsic or prerenal. Urine routine analysis would also help in this regard. The meagre degree of creatinine elevation raises doubt about the possibility of rhabdomyolysis. Creatinine level is considerably elevated out of proportion to BUN due to excessive leak of creatine from damage of muscle cells.

Viral illness may cause dark urine due to hemoglobinuria as well seen in G-6PD deficiency, autoimmune haemolytic anaemia (AIHA) [3,4]. Hepatitis A virus has been found to cause intravascular hemolysis in G-6PD deficiency [5]. Severe hemoglobinuria in consequence to virus A with normal G-6PD status has been observed due to AIHA [6].

So, it appears from the present case report that the diagnostic possibility of Influenza-B virus induced hemoglobinuria associated with viral myositis leading to ARF caused by either G-6PD deficiency or AIHA remains until necessary appropriate laboratory tests are carried out.

**JB Ghosh**

*Consultant Pediatrician  
Ushashi Housing Society  
245 Vivekananda Road  
Kolkata 700 006, West Bengal, India.  
jbgghosh@yahoo.com*

#### REFERENCES

1. Hung PL, Lin PC, Tseng PL. Influenza-B Associated Rhabdomyolysis and Acute Renal Failure. *Indian Pediatr.* 2013;50:595-6.
2. Beetham R. Biochemical investigation of suspected rhabdomyolysis. *Ann Clin Biochem.* 2000;37:581-7
3. Segel GB, Packman CH. Hemolytic Anaemias Resulting from Extracellular Factors-Immune Hemolytic Anaemias. *In: Kilegman RM, Stanton BF, Schor NF, St. Gene JW, Berhman RE, Editors. Nelson Textbook of Pediatrics.* 19<sup>th</sup> ed. Philadelphia. Elsevier; 2011. p. 1680-3.
4. Segel GB, Hackney LR. Glucose-6-Phosphate Dehydrogenase deficiency and Related deficiencies. *In: Kilegman RM, Stanton BF, Schor NF, St. Gene JW, Berhman RE, Editors. Nelson Textbook of Pediatrics.* 19<sup>th</sup> ed. Philadelphia. Elsevier; 2011. p. 1678-80.
5. Sarkar S, Prakash D, Marwah RK. Acute intravascular hemolysis in G-6-P-D deficiency. *Ann Trop Padiatr.* 1993;13:391-4.
6. Lyons DJ, Gilvarry JM, Fielding JF. Severe hemolysis associated with hepatitis A and normal glucose-6-phosphate dehydrogenase status. *Gut.* 1990;31:838-9.

#### REPLY

We are thankful to the author for his interest in our publication. Myoglobin is rapidly and unpredictably eliminated by hepatic metabolism. Therefore, tests for myoglobin in plasma or urine are not a sensitive diagnostic procedure. Red discoloration of the urine when erythrocytes cannot be detected by microscopy must be due to hemoglobinuria or myoglobinuria. The urine microscopic examination showed RBC 0-1/hpf should rule out hematuria.

The enzyme CK is ubiquitously present in striated muscle. When muscle cells disintegrate, CK is released into the bloodstream. Because overall degradation and removal are slow, the concentration of CK remains elevated much longer and in a more consistent manner than that of myoglobin. Consequently, CK is more reliable than myoglobin in assessing the presence and intensity of damage to the muscles. The high level of CK was observed on this case so rhabdomyolysis leading to myoglobinuria should be highly suspected.

The serum creatinine level 3.46 mg/dL and serum urea nitrogen (BUN) 74 mg/dL were studied on the next day after acute renal failure happened. The ratio of BUN and serum creatinine was at borderline for diagnosis of intrinsic ARF. According to past history of this patient, ARF caused by either G-6PD deficiency or AIHA were ruled out.

**PI-LIEN HUNG**

*plhung@vghks.gov.tw*