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.
jbghosh@yahoo.com

REFERENCES

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