Incidentally Detected Elevated Liver Enzymes: From Liver to Muscle

We describe 8 children – with incidentally detected isolated elevation of liver enzymes aspartate aminotransferase and alanine aminotransferase – who were extensively evaluated for hepatic causes before finally being diagnosed to have muscular dystrophy. Serum creatinine phosphokinase levels, if performed early during the work-up, may help in diagnosis of muscle disease and avoid unnecessary investigations for liver disease.

**Keywords:** Anicteric hepatitis, Creatinine phosphokinase, Muscular dystrophy.

Elevated levels of serum aspartate aminotransferase (AST) and serum alanine aminotransferase (ALT) usually indicates hepatocyte injury. However, due to their widespread distribution in the body, serum levels of these enzymes can be elevated in other conditions as well [1,2]. Muscle disorders like muscular dystrophies, inflammatory myopathies and metabolic myopathies can lead to elevated blood levels of creatine phosphokinase (CPK), lactate dehydrogenase (LDH), ALT and AST [1].

Over a period of 6 years, eight boys with a median (range) age of 4.5 (0.5,13) year were referred to us for evaluation of persistently abnormal liver function test (LFT). Abnormal LFTs were detected incidentally and the estimation will help in understanding the complex interplay of dengue infection and immunity.

**Acknowledgments:** Dr Guruprasad R Medigeshi (Associate Professor, THSTI, Faridabad) for support and guidance in research on dengue infection, and Ms Kalaivani M (Department of Biostatistics, AIIMS, New Delhi) for assistance in statistical analysis.

**Contributors:** AS, SKK, RL: involved in study design, implementation and interpretation of data and in the writing of manuscript; RS, MS: involved in study design, implementation and the writing of manuscript; RL: act as guarantor for this paper.

**Funding:** None; **Competing interest:** None stated.

**REFERENCES**

indications for ordering LFT were antituberculous drug therapy ($n=3$), anticonvulsant therapy ($n=1$), evaluation of poor weight gain ($n=3$) and abdominal pain ($n=1$). On detailed evaluation, there was no history or clinical evidence of icterus, organomegaly or other signs to suggest chronic liver disease. The only abnormality in all the serial LFTs was elevated serum transaminases (ALT and AST) in the presence of normal bilirubin, albumin, prothrombin time and other liver enzymes. Mean (range) AST was 320 (134, 550) IU/L and ALT was 342 (154, 560) IU/L. Ultrasonography of abdomen, HbsAg and anti-HCV antibodies were performed in all patients. Five patients had been worked-up for Wilson’s disease and autoimmune hepatitis. Serum ammonia and lactate was done in three patients and celiac serology in one patient. Liver biopsy was performed in three children. All these tests were reported normal.

One child (13-year-old) had a waddling gait. The other seven children had a normal gait but a positive early Gower’s sign. In two of these children, a positive Gower’s sign was seen only on asking the child to get up from the supine position (need to turn on their sides to get up because of weakness of neck flexors - initial component of Gower’s). Mean (range) serum CPK levels were 18,250 (7340, 53617) IU/L. Genetic analysis confirmed a diagnosis of Duchenne muscular dystrophy in seven patients, and one had sarcoglycanopathy on muscle biopsy.

Though many conditions such as viral hepatitis, Wilson’s disease, autoimmune disease, non-alcoholic steatitic hepatitis (NASH) and celiac disease can cause anicteric hepatitis, muscle disease should also be considered early in the differential diagnosis of a child with isolated elevation of AST and ALT. Recognition of muscle disease in children may sometimes be difficult, especially in the pre-symptomatic period [3], and failure to recognize it as a cause of abnormal LFT may lead to unnecessary and invasive investigations like liver biopsy [1,4]. We recommend that serum CPK levels should be performed early in evaluation of children with isolated elevated liver enzymes with no clinical signs/symptoms of liver disease.

Contributors: All authors were involved in acquisition, analysis and interpretation of data, drafting the work and final approval; and are accountable for all aspects of the work

Funding: None; Competing interest: None stated.

*PADMA BALAJI #SRINIVAS SANKARANARAYAN, VISWANATHAN VENKATARAMAN AND #VS SANKARANARAYAN

From Department of Pediatric Neurology and Pediatric Gastroenterology, CHILDS Trust Medical Research foundation and Kanchi Kamakoti CHILDS Trust Hospital, Chennai, India.

*padmabalaji@rocketmail.com

REFERENCES