Role of soluble transferrin receptors study in hepatocellular carcinoma with underlying cirrhosis

Dear Editor,

The measurement of soluble transferrin receptor (StfR) has been proposed as a novel approach to diagnose iron deficiency.[1,2] Soluble transferrin receptor concentration reflects the functional iron status of the body and the rate of erythropoiesis in the bone marrow.[2] Recent reports showed elevation of StfR was observed in malignancies like lymphoma, and hepatocellular carcinoma (HCC).[3,4] They proposed that like other tumor markers, StfR might be synthesized and secreted by hepatocellular carcinoma. We studied the role of StfR in patients with HCC with underlying cirrhosis liver.

A total of 35 patients with HCC with underlying cirrhosis liver (cases) and 20 patients with cirrhosis of liver without HCC (controls), were included in the study. Those patients with hemoglobin less than 10 g/dl and gastrointestinal bleeding in last 3 months were excluded. All patients underwent assays for α fetoprotein (AFP) and StfR.

Diagnosis of HCC was made by Barcelona clinic liver cancer criteria.[5] Serum StfR assay (reference range: 0.9–2.3 ng/ml) was performed using a commercially available kit based on polyclonal antibodies in a sandwich enzyme-linked immunosorbent assay (Orion Diagnostica, Finland) and value of more than 2.3 ng/ml was considered as elevated level. Sensitivity, specificity, positive and negative predictive values were calculated by using the diagnostic test evaluator.

A total of 55 patients were studied, 35 cases and 20 controls. Thirty-five (100%) were males in cases and 18 (90%) were in controls. Mean age was 60.2 years (range: 56–71 years) in cases and 59 years (range: 52–64 years) in control group. Alcohol was the most common etiological factor in cases and control–26 (74%) and 14 (70%), respectively. Histological diagnosis of HCC was made in 9 (30%) of patients. Soluble transferrin receptor level was elevated in 26 (74%) patients with HCC and 5 (25%) patients without HCC (P = 0.003, c2 test). Nineteen (54%) patients had HCC less than 3 cm in diameter. The sensitivity and specificity of StfR were 74 and 65%, respectively. Positive and negative predictive values of StfR as a diagnostic marker in HCC were 84 and 63%, respectively. Mean level of StfR in HCC was 6.2 ng/ml (range: 1.98–7.9 ng/ml) in contrast to 1.8 ng/ml (range: 1.2–3.0 ng/ml) in patients without HCC. α–Fetoprotein level more than 400 ng/ml was present only in 10 (28.5%) patients with HCC in contrast to none among the controls. All ten patients who had AFP more than 400 ng/dl had elevated StfR level.

Since majority of our patients were alcoholics and had advanced liver disease (child C cirrhosis), severity of liver disease, etiology of cirrhosis and level of StfR could not be correlated.

Our results showed 26 (74%) patients out of 35 had elevated StfR level; probably StfR is synthesized and secreted by tumor. In our patients elevation of StfR, unlike AFP, was minimum (maximum three and half times than normal). Elevated StfR and AFP >400 ng/dl were observed in 19 (54%) and 5 (14%) patients with tumor size less than 3 cm. A 3 cm HCC tumor is not hard to diagnose by conventional imaging, thus StfR is unlikely to help to diagnose small HCC.

Thus, preliminary results showed elevated StfR levels were observed in patients with HCC with underlying cirrhosis liver, however it has low sensitivity and specificity to diagnosed HCC.

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References