Cardiohepatic interactions in heart failure

Kalp yetersizliğinde kardiyohepatik etkileşim

To the Editor,

Heart failure (HF) is a fatal and progressive disease, driven by cardiac dysfunction (1). The syndrome of HF is characterized by organ cross-talks, since, heart is central to hemodynamics of many organs both in the form of distributing the oxygenated blood and delivering deoxygenated blood in order to send it to lungs. Among many organ cross-talks in the syndrome of HF, interaction between heart and kidney is relatively well established and defined as “cardiorenal syndrome” (2). Hepatic involvement in the form of cardiohepatic interaction has also been described in patients with chronic HF (3, 4).

In the recent analysis of the SURVIVE database (5), cardiohepatic dysfunction was present in about a half of this cohort of patients with diastolic dysfunction was present in about a half of this cohort of patients with chronic HF (3, 4).

In conclusion, two discrete profiles of cardiohepatic interaction, identified in the study, seem to be critically important targets in order for physicians to tailor the therapy of patients with ADHF.

Figure 1. Hepatic microstructure

Duration of infusion is often short but, sometimes prolonged therapy may be necessary (2). It has been reported that mostly observed complications of long term PGE1 therapy are cortical hyperostosis (CH), gastric outlet obstruction, fluid electrolyte disturbances, and platelet dysfunction (3-5).

In this retrospective case series, 21 newborns with duct-dependent CHD and received PGE1 infusion for longer than 2 weeks were evaluated (Table 1). The mean birth weight and gestational age of the patients were 2982±740 grams and 39.1±2.1 weeks, respectively. The median age of initial PGE1 infusion was three days (1-17). The mean initial dose of PGE1 was 0.022±0.05 mcg/kg/min, and modified accordingly to keep the oxygen saturation above 75%. Average and cumulative dose during treatment were 0.026±0.09 mcg/kg/min and 2219±567 mcg/kg, respectively. The median (min-max) length of the PGE1 therapy was 28 (17-115) days.

Observed complications during long-term PGE1 therapy were noted. The signs of gastric outlet obstruction developed in two patients; at 1st case, on 29th day of therapy (cumulative dose of 3474 mcg) and at 2nd case, on 32nd day of therapy (cumulative dose: 4285 mcg). Ultrasonography (USG) showed elongation of the antpyloric channel with increase in wall thickening (Fig. 1). Hypokalemia (serum K level <3.5 mEq/L) developed in 12 patients on 14-25 days of PGE1 therapy. Three patients on additional furosemide therapy had more prominent hypokalemia. Marked hypokalemia (serum K 1.9-2.7 mEq/L) and metabolic alkalosis (bicarbonate concentrations 28-32 mmol/L) developed in four patients with PGE1 dose of 0.035-0.056 mcg/kg/minute. In another patient, with the PGE1 dose of 0.05 mcg/kg/minute, persistent hypotenremia (serum sodium 120-129 mEq/L) with natriuresis (urine Na: 121.2 mmol/L) were observed after 24 days of PGE1 therapy. Polyuria (8 ml/kg/