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A Case Report of Tumor Lysis Syndrome and Stress Cardiomyopathy after TACE

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Background: Transcatheter Arterial Chemoembolization (TACE) is a chemotherapeutic agent injection method applied directly to tumors, restricting their blood supply. TACE is an established palliative treatment in patients with unresectable HCC that has been shown to improve survival rate. Common adverse complications are Post-TACE syndrome, liver abscess, biliary tract injury and liver failure. Tumor Lysis Syndrome (TLS) usually results from intracellular substances of degrading tumor cells following chemotherapy, manifesting in potentially life-threatening metabolic complications and acute renal failure. As TLS rarely occurs in the treatment of Hepatocellular Carcinoma (HCC), few cases have been reported. We report a case of TLS and stress cardiomyopathy subsequent to TACE. **Case:** A 74-year-old male subject, having undergone coronary angioplasty 10 year previous, and having a history of atrial fibrillation, hypertension and diabetes mellitus, was recently diagnosed with primary huge HCC (9.9×13.5×15 cm), and admitted to hospital for TACE. TACE was performed upon admission. After three days, the patient had RUQ pain, dyspnea and oliguria. Hypocalcaemia (6.6 mg/dL), hyperphosphatemia (6.6 mg/dL), creatinine elevation (2.78 mg/dL) and metabolic acidosis were checked, and critical tumor lysis syndrome clinically diagnosed. As CK-MB (21.5 ng/mL) and Troponin I (10.05 ng/mL) levels were elevated, and cardiologist-performed echocardiography results showed apical ballooning, a diagnosis of stress cardiomyopathy followed. Following ventilator care and hemodialysis, renal function returned, and echocardiography results showed improvement before discharge. Subsequently, the subject has been the object of close observation at our outpatient clinic.

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Hyponatremia in patients with GI bleeding due to portal hypertension treated with terlipressin

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Background: Terlipressin is an analogue of vasopressin used as a vasoactive drug and is highly effective in the treatment of variceal bleeding in patients with portal hypertension. Because terlipressin is also a partial agonist of renal vasopressin V2 receptors, the treatment with terlipressin can be associated with hyponatremia. **Objectives:** To investigate the factors that increase susceptibility to hyponatremia in patients treated with terlipressin for gastrointestinal (GI) bleeding due to portal hypertension. **Methods:** Between January 2007 and March 2012, 85 consecutive patients with the development of hyponatremia after administration of terlipressin for portal-hypertensive bleeding were investigated. And 127 corresponding patients without development of hyponatremia were matched to these patients. **Results:** In the hyponatremic population, serum sodium decreased from 137.2±5.1 mEq/L to 125.3±6.9 mEq/L. The thirty-five patients (42.1%) had a decrease in serum sodium ≤ 10 mEq/L during treatment: in 38 patients (45.8%), between 10 mEq/L and 20 mEq/L and in 10 patients (12%), greater than 20 mEq/L. In multivariate analysis with Logistic regression, the reduction in serum sodium was related to amount of transfused packed red blood cell and MELD score. Patients with low MELD score (OR 0.877 [0.801-0.960]) and small amount of transfused packed red blood cell (OR 0.844 [0.723-0.986]) had the highest risk of hyponatremia. The amount of infused free water and the cumulative dose of terlipressin had not statistical significance. **Conclusion:** The severity of hyponatremia in patients treated with terlipressin for gastrointestinal bleeding due to portal hypertension is diverse, and usually reversible after terlipressin withdrawal. In severe cases, 10 patients (12%) showed decrease in serum sodium over 20 mEq/L, and severe hyponatremia was related to high mortality and morbidity. In multivariate analysis, the reduction in serum sodium was related to amount of transfused packed red blood cell and MELD score.