■ S-225 ■

A Case of Portopulmonary Hypertension Associated with Liver cirrhosis

가천의과대학 길병원 내과

*김의주, 신미승, 오경용, 김명건, 신권철, 박예민, 정욱진, 안태훈, 최인석, 신익균

Many patients with end-stage liver cirrhosis complain of dyspnea or dyspnea on exertion. There are various causes which a clinician should consider in such patients. Although portopulmonary hypertension is an uncommon complication of chronic liver disease or liver cirrhosis, it is a disorder which a clinician should not ignore because the treatment is unique. A 40-year-old female patient who had been diagnosed with liver cirrhosis and portal hypertension due to chronic hepatitis B and followed in the gastroenterology department was admitted to the cardiology department for evaluation of dyspnea on exertion, which had recently worsened . Echocardiography revealed severe pulmonary hypertension (RVsP, 117 mmHg; mPAP, 54.05 mmHg), RA enlargement (d, 50.9 mm) with RVH (Qp/Qs 1.2), and a D-shaped LV with an EF of 86% . A V/Q scan, pulmonary CT angiography, and a lower extremity CT angiography showed no evidence of a V/Q mismatch on the scan and no evidence of a thromboembolism on pulmonary CT angiography and lower extremity CT angiography. The D-dimer was 0.4 μg/mL. The cardiac catheterization showed no detectable shunt. Antinuclear antibody, rheumatoid factor, anti ds-DNA antibody, anti-cardiolipin antibody (IgG and IgM), lupus anticoagulant and anti-mitochondrial, anti-smooth muscle, and anti-LKM antibodies were all negative. Portopulmonary hypertension was the final diagnosis. Beraprost was prescribed and the dyspnea improved. A 2-month follow-up echocardiography showed that pulmonary hypertension had improved (RVsP, 46 mmHg; mPAP, 57 mmHg).

■ S-226 ■

Subarachnoid Hemorrhage after Follow-up Coronary Angiography in Patients with Acute Myocardial Infarction

[1]Heart Center, Chung-Ang University Hospital, [2]Department of Radiology, Chung-Ang University Hospital,
[3]Department of Neurosurgery, Chung-Ang University Hospital, Seoul, Korea

*Min Seok Oh[1], Sang Wook Kim[1], Jeong Eun Kim[1], Eun Jung Cho[1], Jee Eun Kwon[1], Joon Hwa Hong[1], Jae Seung Seo[2], Wang-Soo Lee[1], Jun Soo Byun[2], Kwang Je Lee[1], Tae Ho Kim[1], Chee Jeong Kim[1], Dae Yun Cho[1], Wang Seong Ryu[1], Kyung Tae Kim[3]

Subarachnoid hemorrhage (SAH) is one of the major complication that rarely occurs after coronary angiography (CAG). Acute SAH can be confused with extravasation of contrast media (CM) by disruption of blood-brain barrier on brain computed tomography (CT). We report a patient who developed SAH just after CAG with non-ionic CM and minimal dose of heparin. 55 year old man with history of acute ST elevation myocardial infarction (STEMI) treated with primary percutaneous coronary intervention admitted for follow-up CAG. It was performed by transradial approach using 1,000 U of unfractionated heparin for luminal coating and 70 mL of iodixanol (Visipaque®, GE Healthcare, Ireland) was used for CAG. At the end of procedure, he complained nausea, and rapidly became stuporous. Cranial CT without enhancement showed diffusely increased hounsfield unit (HU) in cisternal space suggesting acute SAH (Fig. 1). The maximal HU was 65 in cisternal space on CT scan. No vascular aneurysm or malformation was detected on cerebral angiography. He partially recovered in mental state and motor weakness after 2 days. SAH was improved on follow-up CT at 2nd hospital day (Fig. 2). Two weeks later, subacute SAH was evident by brain magnetic resonance imaging. He was discharged at 28th hospital day after admission. Only mild sensory impairment was remained on right arm after 12 months. Although low dose heparin is used, acute SAH is a rare but forthcoming complication after CAG.

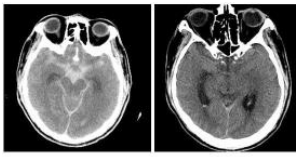

Figure 1. Figure 2.