

PULMONARY INJURY AFTER BALLOON PULMONARY ANGIOPLASTY IN PATIENTS WITH CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION COULD BE ELIMINATED BY AVOIDING VASCULAR INJURY.

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【Background】

Recently, we reported the efficacy of balloon pulmonary angioplasty (BPA) for inoperable patients with chronic thromboembolic pulmonary hypertension (CTEPH). In order to establish BPA as a treatment option for CTEPH, diminishment of the procedural complications should be necessary. Radiographic opacity with worsening of hypoxemia after BPA has been called “reperfusion pulmonary edema” in previous reports and recognized as most serious complication after BPA. Previous studies indicated that pre-procedural severity of pulmonary hypertension had related to the occurrence of this complication and thus, some investigators including us believed that it would be caused by reperfusion itself. However, attempts to eliminate the “reperfusion pulmonary edema” by limiting the size of reperfusion area have not succeeded completely. We hypothesized that vascular injury caused by balloon, wire and contrast media would be the essential cause of pulmonary injury.

【Objective】

To eliminate “reperfusion pulmonary edema” by avoiding pulmonary vascular injury without limiting the size of reperfusion area.

【Method】 We enrolled 15 inoperable patients with CTEPH performed initial procedure of BPA between September 2013 and February 2014. To prevent pulmonary vascular injury, we re-refined intravascular ultra sound guided BPA. During fluoroscopy time up to 60 minutes, we treated as many lesions as possible. Hemodynamic improvements and occurrence of complication after BPA were examined.

【Result】

Pre-procedural mean pulmonary artery pressure (PAP), pulmonary vascular resistance (PVR) and cardiac index (CI) were 41.6 ± 8.9 mmHg, 11.3 ± 5.8 Wood Unit and 2.23 ± 0.8 L/min/m², respectively. The number of treated pulmonary segments and treated lesions in each patient was ranged 2-6 (median 3) and 2-7 (median 5), respectively. Immediate

after the end of procedure, mean PAP was significantly decreased to 35.5 ± 8.9 mmHg and CI was significantly increased to 2.91 ± 0.42 L/min/m². None of the patients experienced occurrence of radiographic opacity with worsening of hypoxemia after BPA. These results indicated that the size of reperfusion area and pre-procedural severity of pulmonary hypertension was not related to the occurrence of pulmonary injury after BPA.

【Conclusion】

The essential cause of pulmonary injury after BPA was not the reperfusion itself but the vascular injury caused by procedure. Therefore, pulmonary injury after BPA would be eliminated by simply avoiding vascular injury during BPA.