Abstract: 95

Terlipressin increases systemic and lowers pulmonary arterial pressure in experimental acute pulmonary embolism

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Background: Patients suffering from high-risk pulmonary embolism (PE) are challenged by increased pulmonary arterial pressure (mPAP) and systemic hypotension

Purpose: We aimed to investigate if terlipressin induces systemic vasoconstriction and pulmonary vasodilation in a porcine model of acute PE.

Methods: Three large autologous PEs were administered to 12 anaesthetized pigs (60 kg). Animals then received four increasing clinical equivalent concentrations of either terlipressin (n=6) or vehicle (n=6). The effects were evaluated in vivo at baseline, after PE and after each concentration by invasive hemodynamics and blood analysis. Isolated pulmonary arteries were evaluated ex vivo in a myograph.

Results: PE caused a 4-fold increase in pulmonary vascular resistance (PVR) compared to baseline (Figure 1). Terlipressin caused an increase in mean systemic blood pressure (+28±5 mmHg, p<0.0001) and systemic vascular resistance (Figure 1) compared to vehicle. Contrary, terlipressin caused a decrease in mPAP (-6.5±1.8 mmHg, p=0.005) and a trend towards a decrease in PVR (Figure 1). Ex vivo, terlipressin caused relaxation of pulmonary arteries (17±4 %, p=0.0007). Terlipressin caused a decrease in cardiac output (-2.5±0.5 L/min, p<0.0001), and an increase in plasma lactate (+2.7±0.2 mmol/L, p<0.0001), indicating systemic hypoperfusion. S100b, a biomarker of cerebral ischemia, remained unchanged, suggesting preserved cerebral perfusion (+0.17±0.11 µg/l, p=0.51).

Conclusion: Terlipressin caused systemic vasoconstriction and pulmonary vasorelaxation in a porcine model of acute PE. As a net effect, cardiac output declined probably due to a predominant systemic vasoconstrictor effect of terlipressin.
A: Systemic Vascular Resistance (SVR)

B: Pulmonary Vascular Resistance (PVR)

Data presented as mean ± SEM. * = p < 0.05, ** = p < 0.01, *** = p < 0.001, **** = p < 0.0001, compared to vehicle.