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Cardio-pulmonary consequences of thoracic irradiation in rats

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The risk of early radiation pneumonitis limits the radiation dose and efficacy of radiotherapy of thoracic tumours. Besides lung dose, in rats (1) and patients (2) dose to the heart was shown to be a risk factor of radiation pneumonitis. Here we investigated whether the enhanced damage caused by combined heart and lung irradiation can be understood from their individual effects on cardio-pulmonary physiology.
First we investigated whether the enhanced increase in respiratory rate observed after combined heart and lung irradiation, as compared to irradiation of the lung alone is reflected in the classical signs of radiation pneumonitis. Indeed, we showed that heart irradiation enhanced inflammation and fibrosis.
Next we investigated whether early changes in heart function could be observed during the peak of radiation pneumonitis. Although the heart is regarded as a late responding tissue, we did observe reduced FDG uptake and perivascular fibrosis 8 weeks after irradiation. Moreover, we observed increased left ventricle (LV) end-diastolic pressure and relaxation time, both correlating excellently with the perivascular fibrosis of the LV. Altogether this indicates that heart irradiation induces early LV diastolic dysfunction.
Lung irradiation alone resulted in irradiated-volume dependent pulmonary vascular remodelling leading to increased pulmonary artery pressure and pulmonary perivascular oedema (3). Moreover, we observed an increase in relaxation time of the LV, suggesting also lung irradiation impairs LV diastolic function.
Taken together these results indicate that heart and lung irradiation both induce loss of left ventricle diastolic function albeit through different mechanisms. In agreement with the above we found that heart irradiation leads to enhanced levels of interstitial oedema, whereas both heart and lung irradiation induce perivascular oedema as a known consequence of diastolic dysfunction. Our results explain the enhanced inflammation and fibrosis observed after combined lung and heart irradiation.
In summary, heart and lung irradiation independently impair cardiac diastolic performance. In our rat model heart irradiation induces early subclinical patho-physiological changes that, if combined with lung irradiation may manifest as an enhanced risk and severity of radiation pneumonitis.

1. van Luijk P et al., Cancer Res. 2005.
2. Huang EX et al., Acta Oncol. 2011.
3. Ghobadi G et al., Thorax 2012 .

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