Oesophagopericardial fistula as a late complication of stereotactic radiotherapy for recurrent ventricular tachycardia

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Stereotactic body radiotherapy (SBRT) has been suggested as a promising therapeutic alternative in cases of failed catheter ablation for recurrent ventricular tachycardias (VTs).¹ Early results triggered a wave of enthusiasm, while severe adverse effects have been reported only in an abstract form (Robinson CG, et al. Int | Radiat Oncol 2019;105:682).

A 67-year-old patient with a history of inferior myocardial infarction and coronary artery bypass grafting using the gastroepiploic artery was implanted with Cardioverter-Implantable Defibrilator (ICD) for recurrences of VT 16 years later. He underwent catheter ablation in an expert centre, tarthree different geting morphologies of VT from the inferior wall. Despite noninducibility at the end, the patient had recurrences of two faster VTs 2 years later. Stereotactic body radiotherwas performed (CyberKnife, Accuray), based on electroanatomical maps positron emission tomography/computed to-

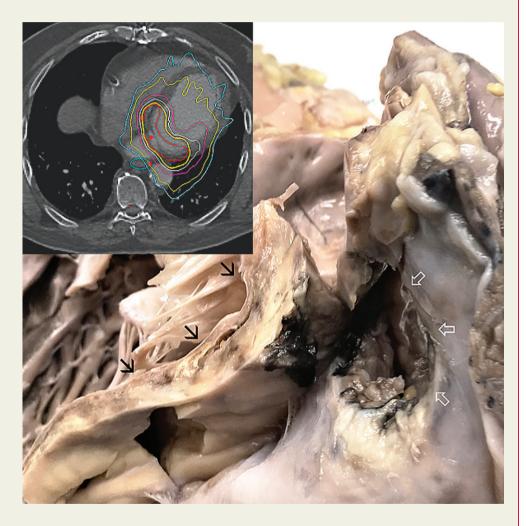


Figure 1 Post-mortem macroscopic picture of the myocardial substrate in the inferior wall (black arrows) and adjacent oesophagopericardial fistula opening through the parietal pericardium (open arrows). *Inset*: CT angiogram depicting radiosurgical treatment plan with isodose lines.

mography (CT) identification of the scar (25 Gy on 76% isodose, target volume 70 cm 3 , CTV covered entire scar, Clinical and Planning Target Volume (CTV-PTV) margin was isotropic 3 mm) (*Figure 1*). The maximum dose to oesophagus met recommended dose–volume constraints for oesophagus [D5 mL = 9.23 Gy, Dnear max (0.035 ccm) = 13.9 Gy, Dmax = 14.46 Gy]. We used 162 non-coplanar beams instead of arcs. The patient presented with dysphagia and early oesophagitis was confirmed by endoscopy 18 days later. All complaints

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resolved on a high dose of antiulcer therapy and the patient was asymptomatic, without VT recurrences. Six months later, he was admitted for dysphagia and was diagnosed with a large deep ulcer in the terminal oesophagus. After the insertion of percutaneous endoscopic gastrostomy and antiulcer therapy, the patient underwent contrast oesophagography with no signs of perforation. He was readmitted 3 months from the beginning of symptoms for progression of dysphagia and haematemesis. Endoscopy revealed necrotic ulcer with venous bleeding, unsuitable for any local intervention. On CT, a cavity around the distal oesophagus with air was documented. Corrective surgery was considered high risk. A few days later, more severe bleeding occurred leading to hypovolaemic shock and melena. After volume expansion and repeated cardiopulmonary resuscitation for electromechanical dissociation, the patient presented with episodes of fast VT. After transient stabilization with antiarrhythmics, the shock progressed and the patient died with terminal asystole. A post-mortem examination confirmed oesophagopericardial fistula (*Figure 1*).

Our case illustrates that SBRT may be complicated even in a long-term course. This is well known from oncology literature on cardiac toxicity after higher dose chest radiotherapy.² Oesophageal injury was described 5–40 months after radiotherapy.³ In our patient, previous bypass surgery with gastroepiploic artery might contribute to oesophageal damage. The lessons for planning SBRT may be to consider decreasing the prescribed dose and/or lower coverage with prescribed isodose whenever the PTV is larger or an additional risk factor is present. The risk/benefit of SBRT for VT should be always carefully considered and long-term follow-up is advisable.

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