Simon Tang
1 Campbell Street Liverpool 2170

Ingham Institute for Applied Medical Research

Research Fellow

1. Ingham Institute of Applied Medical Research, Liverpool, NSW, Australia
2. Cancer Therapy Centre, Liverpool Hospital, NSW, Australia
3. University of New South Wales, NSW, Australia
4. Department of Cardiology, Liverpool Hospital, NSW, Australia
5. University of Sydney, NSW, Australia
6. Siemens Healthcare Pty Ltd, Sydney, Australia

ABSTRACT

Please type in your abstract up to a MAXIMUM of 500 words. Figures may be included.

Purpose: Crude rates of late cardiotoxicity in oesophageal carcinoma patients are reportedly 10.8%[1]. Subclinical cardiac dysfunction manifested as declines in ejection fraction and perfusion abnormalities too have been documented[2, 3]. This case study aimed to document myocardial tissue changes (by cardiac tissue mapping (MyoMaps) using cardiac MRI (CMR) in a patient treated with oesophageal carcinoma.

Materials & Methods:
A 67 year old gentleman with Stage IB T2N0M0 squamous cell carcinoma of the lower oesophagus was treated with chemoradiation; 50Gy in 25 fractions using a 3D conformal technique with concurrent carboplatin/paclitaxel. This patient underwent 3 separate CMR scans, one prior, 6 weeks, and 12 months following completion of his chemoradiation. A clinically modified Look Locker Inversion (MOLLI) sequence was used to generate myocardial short axis T1 maps (MyoMaps, Siemens), pre and 15 minutes post administration of a gadolinium based contrast agent, as well as T2 maps (MyoMaps, Siemens) at 3 Tesla. T1, T1 post contrast and T2 relaxation times of the LV were acquired with MRI mapping software(cv42, v4.5, Circle Software). Extracellular volume(ECV) was derived from the myocardial partitioning coefficient (λ), adjusting for haematocrit. Values were recorded in the American Heart Association(AHA) 16 Segment Model[4].

Case Study – Utility of cardiac MRI mapping in the quantification of myocardial toxicity following concurrent chemoradiation for oesophageal Carcinoma

AUTHOR(S)
Simon Tang1,2,3, Eng-Siew Koh1,2,3, Robba Rai1,2,3, James Otton1, Mark Lee2,3, David Tran1, Lois Holloway1,2,3,5, Liza Thomas3,5, Benjamin Schmitt6, Gary Liney1,3

1. Ingham Institute of Applied Medical Research, Liverpool, NSW, Australia
2. Cancer Therapy Centre, Liverpool Hospital, NSW, Australia
3. University of New South Wales, NSW, Australia
4. Department of Cardiology, Liverpool Hospital, NSW, Australia
5. University of Sydney, NSW, Australia
6. Siemens Healthcare Pty Ltd, Sydney, Australia
Results:
The mean heart dose was 28.82 Gy. The mean LV dose was 14.16 Gy. Mean dose delivered to the left ventricular segments was heterogeneous, with segments 3 and 4 receiving 30 Gy or more, segments 2 and 5 receiving 20 Gy or more, and segments 6, 10 and 11 receiving 10 Gy or move. The basal segments received the highest doses.

Changes in the T1 values are as illustrated in Figure 1. Visually there appears to be an increase in native T1 values post chemoradiation, most prominently 12 months following treatment, which is occurring most prominently in segments 3, 4 and 5, which correspondingly received the highest radiation doses. A 12 month increase in T2 relaxation time values was also seen, although occurring more globally throughout the left ventricle. The ECV percentage transiently increased 6 weeks following chemoradiation.

Figure 1 - T1 Values

A, B and C represent pre treatment, 6 week post treatment, and 12 months post treatment time points respectively. Elevation of T1 values were most pronounced at 12 months in the basal segments.

Conclusions:
The use of CMR mapping for quantitative assessment of the myocardium following cancer therapy treatment shows promise, and experience with this patient has demonstrated feasibility. In this case study there appears to be an elevation of T1 and T2 relaxation times occurring 12 months following treatment, which is preceded by an increase in ECV percentage immediately following chemoradiation. The use of cardiac MRI mapping may provide novel information regarding acute to sub-acute myocardial changes following radiation therapy.

References