Takotsubo syndrome following 5-Fluorouracil chemotherapy for oesophageal cancer: a case report.

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Background:

Cardiotoxicity with 5-Fluorouracil (5-FU) chemotherapy is relatively common at around 4%, predominantly due to coronary vasospasm with a small number of cases of Takotsubo syndrome reported. We report a case of Takotsubo associated with 5-FU in the absence of cardiac risk.

Case report:

A 55 year old lady presented 1 week after her 2nd cycle of FOLFOX (5-FU and Oxaliplatin) chemotherapy with dyspnoea and chest pain.

She was diagnosed with metastatic oesophageal squamous cell carcinoma early 2017 and was receiving first-line palliative chemotherapy. Past medical history was of stage 2b cervical cancer treated radically with combined chemo- and localised radiotherapy. She had no previous cardiac history or risk factors.
Diagnostic imaging revealed an oesophageal tumour with local node involvement, pleural effusions, and rib metastases. She received 36 Gy radiotherapy to the primary lesion and required gastrostomy and oesophageal stent placement for persistent dysphagia.

An ECG showed new inferolateral T wave inversion with a prolonged QTc interval (530 ms). Troponin-I was mildly raised at 46 ng/L (0 – 40) and her BNP was very high (2795 ng/L (<50)). Echocardiogram confirmed apical and mid-apical akinesia with an LVEF of 30%. CT coronary angiogram demonstrated no coronary atheroma.

Findings were consistent with Takotsubo syndrome with the likely causative agent being 5-FU. Carvedilol 6.25mg daily was initiated and uptitrated as tolerated with symptomatic improvement. At 1 month, her QTc interval had corrected to 380 ms and LVEF recovered to 57% with no persisting regional wall motion abnormality. 5-FU was changed to Ralitrexed and she completed a further 4 cycles of treatment without complication.

Discussion:

Patients receiving chemotherapy are at risk of significant cardiotoxicity and should be closely monitored. Takotsubo syndrome is a rare but important toxicity caused by catecholamine release with resulting transient regional myocardial dysfunction. ECG findings of acquired QTc prolongation with deep T wave inversion in the context of apical akinesia and reduced LVEF are diagnostic in this case. Patients may not have cardiac risk factors. A possible paraneoplastic effect could render the myocardium susceptible to cardiotoxic chemotherapy. Further research into causality is warranted.