

Inflammatory Bowel Disease & Myocarditis:

T1-Mapping the heart of the problem

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Competing Interests

EJC, SGM and VMF have no competing interests to declare.

SKP has patent authorship rights for U.S. patent 9285446 B2. Systems and methods for shortened look locker inversion recovery (Sh-MOLLI) cardiac gated mapping of T1. Granted March 15, 2016. All rights transferred to Siemens Medical.

Inflammatory bowel disease (IBD) may be associated with a number of extra-intestinal complications, including, rarely, myocarditis. Diagnosis of this can be difficult however, but we highlight the utility of cardiovascular magnetic resonance (CMR) in this patient group.

Two hospitalised patients with IBD (one with an exacerbation of ulcerative colitis, and one on a tapering schedule of steroid treatment for Crohn's Disease) had experienced crushing chest pain, a troponin rise and had normal coronary arteries on angiography. Both patients underwent CMR at 1.5 Tesla, including dark-blood T2-weighted, late gadolinium enhancement (LGE) imaging, and native T1-mapping (Figure 1). In both cases, global myocardial oedema was not apparent on conventional T2-weighted imaging, with a myocardial-to-skeletal muscle T2 signal intensity ratio of <1.9 .

LGE showed only small areas of mid-wall enhancement in the septum and patchy enhancement in the basal inferolateral wall. However, native T1-mapping revealed a larger extent of myocardial involvement, with marked elevated global myocardial T1 values in both cases (UC: 1166 ± 77 ms; CD: 1047 ± 99 ms; normal ShMOLLI myocardial $T1 = 962 \pm 25$ ms). This was consistent with (although not specific for) acute myocardial oedema ($T1 > 990$ ms).

Both cases demonstrate the importance of recognising that myocarditis can accompany active IBD. CMR can be used to confirm this diagnosis within the clinical context, using novel mapping techniques revealing global myocardial oedema, which may be missed by conventional T2-weighted imaging if the degree of inflammation is subtle, or if skeletal muscle is also inflamed as part of a systemic response. A correct diagnosis

of myocarditis may then be managed appropriately, avoiding treatment for acute coronary syndrome with antiplatelet agents and anticoagulation, which may exacerbate intestinal bleeding in active IBD.

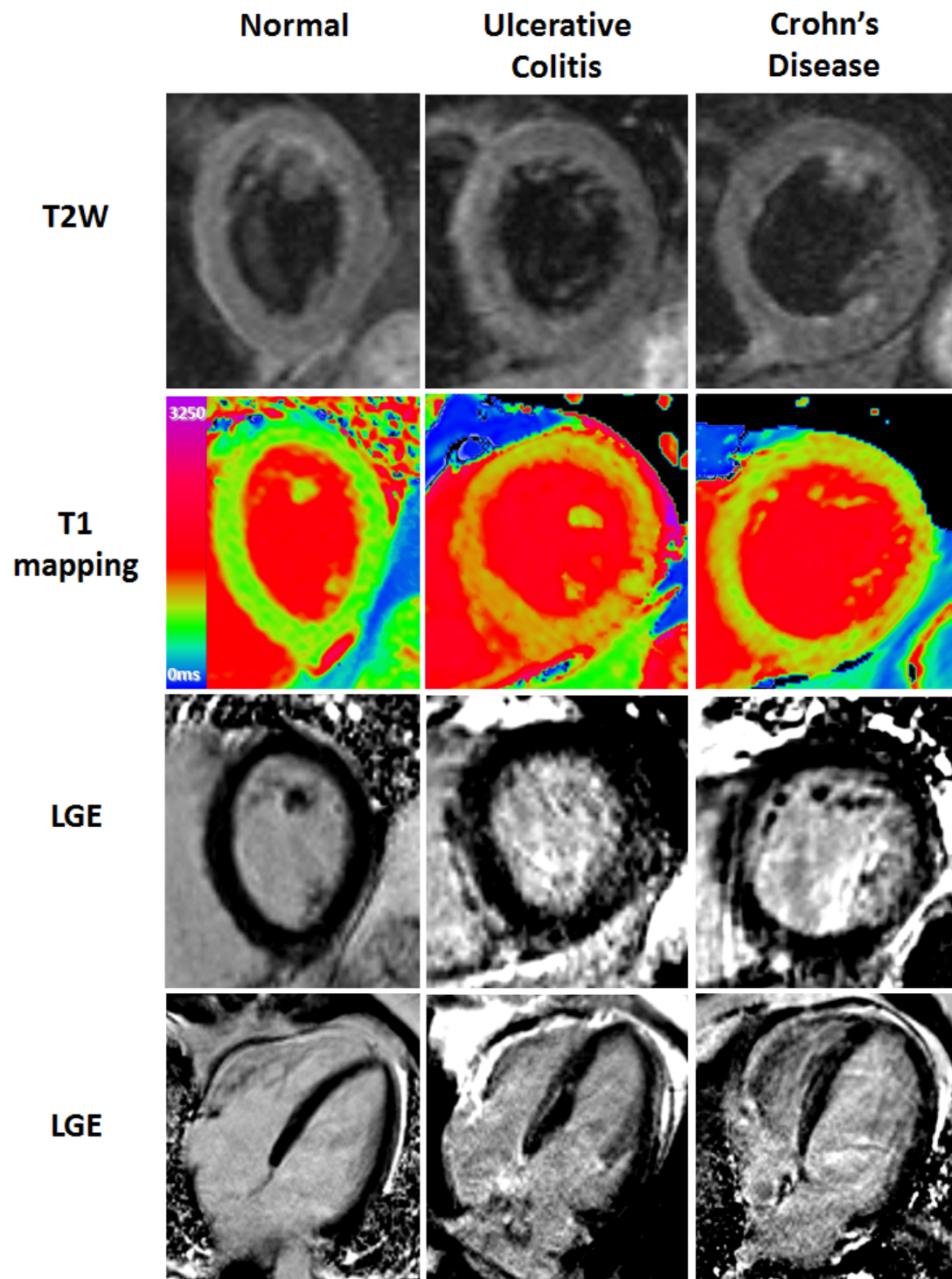


Figure 1: Multiparametric CMR (1.5 Tesla) in a normal control, a patient with ulcerative colitis (UC) and a patient with Crohn's disease (CD).

The left ventricular ejection fraction was 55% in the patient with UC and 30% in the patient with CD. Dark-blood T2-weighted (T2W) imaging showed a normal myocardial to skeletal muscle (not shown) T2 signal intensity ratio of <1.9 in both cases. T1-mapping (ShMOLLI) showed markedly increased myocardial T1 relaxation time in both patients (UC: $1166 \pm 77\text{ms}$ and CD: $1047 \pm 99\text{ms}$; normal ShMOLLI myocardial T1 = $962 \pm 25\text{ms}$ at 1.5T). Late gadolinium enhancement (LGE) showed small areas of mid-wall enhancement in the septum and patchy enhancement in the basal inferolateral wall in both patients, with no myocardial infarction.