assessments of LV function. T2-weighted imaging was conducted for detection of myocardial oedema. Gadolinium late enhancement (LE) imaging was conducted for detection of fibrosis.

Results: Of the 37 patients 14 were male. The mean age was 51.9±2.4 years. 20 patients also underwent coronary angiography, and only one patient had a significant stenosis of a marginal branch. Four patients (11%) had a cardiac MRI scan with pathological findings: three patients (8%) had a non-circumferential PE of ≤ 5mm and an otherwise normal MRI. In 12 patients a PE ranging from 4 to 30mm was associated with other pathological findings. Quantitative analysis of LV function revealed a mean ejection fraction (EF) of 51.9±2.3%. EF was reduced (<55%) in 20 patients (54%). 7 patients (19%) showed hypointense areas in T2-weighted images indicative of myocardial oedema. LE of the myocardium was observed in 23 patients (62%), all demonstrating a non-ischemic pattern of LE or multiple small endocardial LE zones not compatible with myocardial infarction due to coronary artery disease. Lasty, 4 patients (11%) showed the typical pattern of endomyocardial fibrosis with oedema and contrast enhancement of the myocardial/endocardial border zone and thrombotic material at the left and right ventricular apex.

Conclusions: Cardiac MRI revealed pathologies in 90% of the study cohort. Yet, the pattern of cardiac manifestations varies significantly. More than half of the patients showed cardiomypathy with reduced systolic LV function. Non-ischemic LE was present in the majority of patients and combined in one third of cases with myocardial oedema indicating acute inflammation. The unique pattern of endomyocardial fibrosis was observed in 11% of the patients.

3502|BENCH
RNA helicase (2C) inhibitor prevent entero viral-mediated myocardiopathy
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Background: Coxsackievirus B3 (CVB3) is known as an important cause of myocarditis and dilated cardiomyopathy in children and adult. Enterovirus-2C (E2C), a viral RNA helicase, inhibits the host NF-kB activity, which affects to host protein synthesis. We hypothesize that the inhibition of 2C may suppress virus replication and prevent enterovirus-mediated cardiomyopathy.

Methods and results: We generated a chemically modified small molecule structure of entero virus-2C inhibitor (E2CI). From in vitro assay, the strong E2C and control rat, KR22865, was selected for in vivo test which were performed using DBA/2 strain to establish chronic myocarditis. Mice were treated by KR22865 intraperitoneally injection for three consecutive days at a dose 8mg/kg /day after day 3 CVB3 infection (p.i) (n=33) that is similar to human patient antiviral therapy. To obtain susceptible tissue for microscopy, 19 of the 39 patients underwent myocardial biopsy and MRI biopsy. No complications occurred. In 19 of the patients, the MRI histology was diagnostic of sarcoidosis (sensitivity, 100%). Importantly, myocarditis was frequently used as a “rescue” diagnostic strategy since 16 of the 19 patients had a history of either one (10 patients) or several (6 patients) prior negative cardiac biopsies. In the 20 “hot” mLNs patients who did not undergo myocarditis, sarcoidosis had been verified by EMB in 13 patients in 4 repeated attempts, by biopsy of extrathoracic tissue in 6 patients and from an explanted heart in 1 patient.

Conclusion: Cardiac MRI revealed pathologies in 90% of the study cohort. Yet, the pattern of cardiac manifestations varies significantly. More than half of the patients showed cardiomypathy with reduced systolic LV function. Non-ischemic LE was present in the majority of patients and combined in one third of cases with myocardial oedema indicating acute inflammation. The unique pattern of endomyocardial fibrosis was observed in 11% of the patients.