Oral presentation

Open Access Does MRI lend insight into septal enhancement patterns? The significance of conduction defects on EKG

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Background

Interventricular conduction delay is a progressive problem and in patients with heart failure it portents a poor prognosis, especially LBBB. However, the myopathological etiology of IVCD remains poorly understood. Cardiac MRI (CMR) is a very sensitive method to delineate myocardial tissue abnormalities due to inflammation, ischemia, infarction or infiltration, and the relationship of the presence of any abnormal septal delayed hyperenhancement (DHE) to the conduction system may be important.

Hypothesis

We hypothesize that patients with conduction disorders on EKG will uniformly have abnormal myocardial septal DHE by CMR.

Methods

A study of 215 consecutive pts with DHE CMRI performed in the last 18 months and their most proximate EKG was analyzed for QRS duration, LBBB, RBBB and interventricular conduction delay (IVCD) pattern. The DHE image was analyzed for the presence (+) or absence (-) of any basal anterior or anteroseptal enhancement.

Results

Of the 215 patients, 175 patients' EKG's were available within 1 month. Of those, 71 (40%) had a conduction defect on EKG: 19 patients (11%) had LBBB, 25 (14%) had RBBB and 27 (15%) had IVCD. Of those, 11% of the DHE+ pts had LBBB, as did 11% of the DHE- pts (p = NS).

Similarly, 13% of DHE+ pts had RBBB while 16% of DHEpts had RBBB (p = NS). Likewise, 20% of DHE+ pts had IVCD while 11% of DHE- pts had IVCD (p = NS). Fully 50/86 (58%) of DHE+ patients showed some type of EKG abnormality (RBBB, LBBB or IVCD), along with 64/89 (72%) of DHE- also revealing some level of conduction abnormality ($\chi 2 = 3.07$, 1 df, p = .08).

Conclusion

Counterintuitively, despite the high resolution capabilities of CMR, the presence or absence of DHE signal does not influence the EKG conduction pattern, suggesting that infarct patterns affect mechanical but not electrical properties within the heart. The conduction system appears to be relatively 'immune' to disorders typically depicted by resolved infarct imaging. We show for the first time that infarct presence or absence does not materially predict either presence, absence or even type of conduction disorder.