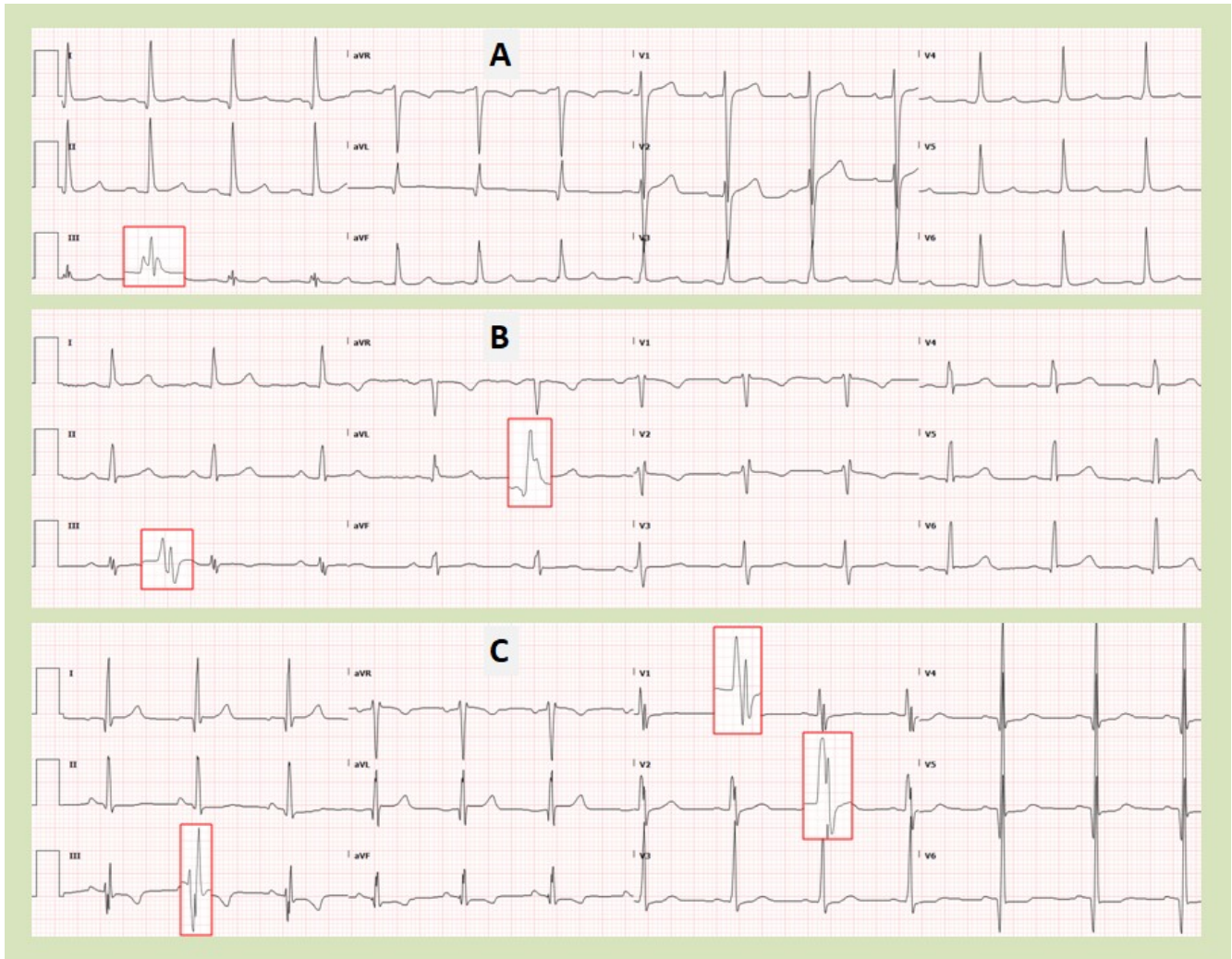


QRS Fragmentation: *Squiggly Curiosities!*

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Description

The above electrocardiograms (ECGs) have variable degrees of QRS fragmentation (inserts). Figure A demonstrates fragmented QRS only in lead III. Figure B fragmentation of the QRS is seen predominantly in Leads III and aVL, with rSr' pattern in leads V1 and V2. Figure C reveals widespread QRS fragmentation, predominantly in leads III, V1 and V2, and to a lesser extent in leads aVL and aVF. The QRS duration in all ECGs is less than 110 milliseconds, and therefore does not meet criteria for an intraventricular conduction delay (IVCD).

All ECGs belong to young patients with sickle cell disease who had previous hospital admissions with chest pain and dyspnea. They all had mild to moderate increase in LV wall thickness measurements by 2-dimensional (2-D) echocardiography. They also all had documentation of either troponin I or NT-proBNP level elevations at some point in their care. They all suffered significant anemia expected of sickle cell disease. None of the patients manifested any specific tachy- or brady- arrhythmia beyond nonspecific isolated ectopy.

Discussion

QRS Fragmentation on the ECG has been known for many decades, previously described as high frequency components attributed to ventricular enlargement and infarction [1]. It has been defined as presence of extra R wave (R') or notching in the S wave nadir in the setting of a narrow QRS complex [2]. It is an important marker of arrhythmia and mortality in several cardiac conditions [3].

Sickle cell patients have higher odds of an abnormal ECG, especially nonspecific ST-T changes, left ventricular hypertrophy, T-wave abnormalities, long QTc interval, and ischemia [4]. They are also prone to having myocardial damage due to repetitive microvascular injury [5], and as a result are likely to manifest QRS fragmentation on ECG, as the three ECGs discussed above demonstrate. This perhaps would be a worthwhile correlation to look into with further studies to help elucidate the prevalence and impact of QRS fragmentation on the risk of ventricular arrhythmia and sudden cardiac death in sickle cell patients [6].

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