CASE REPORT

Transient left septal fascicular block in the setting of acute coronary syndrome associated with giant slurring variant J-wave

Andrés Ricardo Pérez-Riera MD, PhD¹ | Raimundo Barbosa-Barros MD² |
Wallam Lima Aragão MD² | Rodrigo Daminello-Raimundo PhD¹ |
Luiz Carlos de Abreu PhD¹ | Joseane Elza Tonussi Mendes Rossette do Valle MD¹ |
Isabel Cristina Esposito Sorpreso MD¹ | Kjell Nikus MD³

¹Design of Studies and Scientific Writing Laboratory in the ABC School of Medicine, Santo André, São Paulo, Brazil

²Coronary Center of the Messejana Hospital Dr. Carlos Alberto Studart Gomes, Fortaleza, Brazil

³Heart Center, Tampere University Hospital and Faculty of Medicine and Life Sciences, University of Tampere, Tampere, Finland

Correspondence

Andrés Ricardo Pérez-Riera, MD, PhD,
Design of Studies and Scientific Writing
Laboratory in the ABC School of Medicine,
São Paulo-SP, Brazil.
Email: riera@uol.com.br

We report a case of acute coronary syndrome with transient prominent anterior QRS forces (PAF) caused by proximal subocclusion of the left anterior descending (LAD) coronary artery before the first septal perforator branch. The ECG change indicates left septal fascicular block (LSFB) with associated slurring-type giant J-wave. Currently, this J-wave variant is considered as a lambda-like wave or QRS-ST-T “triangulation”. Its presence is indicative of poor prognosis because of the risk for cardiac arrest as a consequence of ventricular tachycardia/ventricular fibrillation (VT/VF).

KEYWORDS

acute coronary syndrome, giant slurring variant J-wave, prominent QRS anterior forces, transient left anterior fascicular block

1 | CASE REPORT

A 68-year-old man, hypertensive smoker, arrived at the hospital complaining of intense oppressive retrosternal pain associated with dyspnea that started 5 hr earlier. The first electrocardiogram (ECG) was performed (Figures 1 and 2) before transfer to a tertiary hospital, where the second ECG was conducted (Figure 3); the patient still had mild chest pain.

2 | PHYSICAL EXAMINATION

Sinus tachycardia, normal heart sounds, no murmurs, blood pressure 150/100 mmHg. Clean lungs.

He was referred to primary percutaneous coronary intervention (PCI), which revealed a subtotal occlusion of the proximal LAD, treated with drug-eluting stent implantation. After the PCI he was hemodynamically stable and pain-free.

3 | DISCUSSION

The first ECG shows transient PAF with several criteria for LSFB. Additionally, during the acute phase of MI, PAF are associated with J-wave slurring, “lambda-like pattern” or monophasic QRS-ST-T complexes, which, in turn are strongly associated with VT/VF (Aizawa et al., 2012).

We and others demonstrated in numerous publications that transient PAF can be caused by critical proximal stenosis of the LAD causing ischemic LSFB (Perez-Riera, Barbosa-Barros, Penachini da Costa de Rezende Barbosa, Daminello-Raimundo, & de Abreu, 2017; Riera et al., 2008).

This ECG pattern reflects ischemia of the left septal fascicle. Myocardial ischemia causes LSFB, which is associated with abnormal augmentation of repolarization dispersion, as indicated by various alterations in the shape and duration of the action potentials (AP) across the left ventricular wall (including triangulated APs); this also frequently triggers re-entry arrhythmias (Behrens, Li, &

FIGURE 1 Sinus rhythm, heart rate 88 bpm, P-wave duration 120 ms, P axis +55°, PR interval 160 ms, prolonged R-wave peak time (RWPT) in V_1 – V_2 , QRS-axis-10°, QRS duration 120 ms. No clear distinction between the end of QRS and the beginning of ST (QRS–ST–T “triangulation”), embryonic q wave in V_3 – V_4 , PAF: R-wave “in crescendo” from V_1 to V_4 and decreasing in V_5 – V_6 , very high J-wave of end-QRS slurring type across all precordial leads, and prolonged QT/QTc interval (500/588 ms). Note: J-wave end-QRS slurring with lambda-like/Gussak-wave (Gussak, Bjerregaard, & Kostis, 2004) or triangular QRS–ST–T waveform. Conclusion: Left atrial enlargement, LSFB (Perez-Riera, de Abreu, Barbosa-Barros, Nikus, & Baranchuk, 2016), and giant slurring variant J-wave end-QRS

FIGURE 2 Schematic figure of J-wave variants: end-QRS notching, end-QRS slurring represented by the present case. Early repolarization with or without ST-segment elevation is characterized by end-QRS notching or slurring (the present case)

Franz, 1997). These QRS–ST–T patterns (“monophasic” and “triangulated”, among others) are most likely not specific for myocardial ischemia. Arrhythmogenic “lambda-like” ST elevation patterns

have been reported in “atypical Brugada syndrome (BrS)” (Riera, Ferreira, Schapachnik, Sanches, & Moffa, 2004) and in coronary artery disease scenarios, such as acute myocardial infarction (AMI)

FIGURE 3 Sinus tachycardia, QRS-axis $+50^\circ$, rS in V_1 and QS in V_2 , ST-segment elevation in V_1 – V_3 followed by negative or biphasic “postischemic” T-wave from V_2 to V_5 . Conclusion: misdeal myocardial infarction (MI), postischemic anterior T waves

complicated with VF (Kukla, Jastrzebski, Sacha, & Bryniarski, 2008). These observations reveal a mechanism that triggers sustained VT/VF during AMI.

The J point marks approximately the end of the QRS and the ST-segment onset. The degree of J-point elevation determines the risk for sudden cardiac death (Tikkanen et al., 2009), and increases in J-wave amplitude often precede VF events (Haissaguerre et al., 2008; Nam, Kim, & Antzelevitch, 2008). QRS-ST-T triangulation is a precursor of sustained VT during acute myocardial ischemia (Batchvarov & Behr, 2015).

A J wave is observed in several channelopathies, such as early repolarization syndrome, BrS, idiopathic VF, and congenital short QT syndrome. Additionally, in hypothermia, severe hypercalcemia, central nervous system injury, such as subarachnoid hemorrhage, myocarditis, cocaine and haloperidol overdose, left ventricular hypertrophy due to hypertension, acute coronary syndromes (Kukla et al., 2008), Prinzmetal angina, Takotsubo cardiomyopathy, after resuscitation from cardiac arrest, and other miscellaneous entities.

The mechanisms underlying the electrocardiographic and arrhythmic manifestations are controversial. Some J-wave etiologies may be related to abnormalities of late *depolarization*, others to early *repolarization* and some to a combination of *depolarization/repolarization* defects (eclectic theory).

4 | CONCLUSION

We report a case of transient LSFB caused by proximal subocclusion of the LAD, treated with stent implantation. Besides the numerous criteria for LSFB, the presence of a high-amplitude J wave with characteristics of the slurring variant stands out. It is very important

for physicians to be aware of this uncommon ECG pattern, where a proximal critical stenosis of the LAD is the underlying cause. The additional presence of a high-amplitude J wave is a known marker of potentially fatal arrhythmias, cardiogenic shock, and high in-hospital mortality.

CONFLICTS OF INTEREST

None.

ORCID

Andrés Ricardo Pérez-Riera <http://orcid.org/0000-0003-4948-538X>

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