ESC European Society of Cardiology
European Heart Journal - Case Reports (2022) **6**, 1
https://doi.org/10.1093/ehjcr/ytac249

Ischaemia-induced J waves

Newly developed or augmented J (Osborn) waves have been shown to be associated with acute myocardial ischaemia occurring in the context of spontaneous or induced coronary artery spasm or acute myocardial infarction.[1–3] Experimentally, it was shown that J wave appearance represents an initial and transient response to acute myocardial ischaemia owing to both conduction delay in the ischaemic myocardium and accentuation of the transient outward potassium current (Ito)-mediated action potential notch during early ventricular repolarization in the epicardium but not the endocardium.[3,4] Ischaemia-induced J waves are registered in electrocardiographic (ECG) leads facing the ischaemic territory.[1–3] Persistence of ischaemia results in complete loss of the epicardial action potential notch leading to a progressively augmented J wave that merges with a steeply downward-sloping elevated ST-segment giving rise a 'triangulated' QRS–ST–T complex resembling the Greek letter 'λ' (lambda).[1,3,4] Furthermore, ischaemia-induced J waves denote the presence of a markedly increased transmural dispersion of ventricular repolarization on the epicardial surface across the ischaemic border, which facilitates the development of Phase II reentry and formation of short-coupled ectopic beats ('R-on-T' phenomenon) capable of initiating ventricular fibrillation (VF).[1,3,4]

Cha *et al.*[5] recently reported the interesting case of a 61-year-old male patient, in whom the J waves that were noted in leads aVL, I and V6 during an episode of recurrent angina resolved after revascularization of a culprit lesion in the proximal left circumflex (LCx) artery, thereby proving their ischaemic origin. Scrutiny of the admission ECG, also reveals a negative J wave in lead aVR, that is a lead facing through the left ventricular (LV) cavity, the apex, and lateral wall, and is directionally opposite to leads I, II, V5, and V6. Furthermore, a 'triangulated' 'lambda-like' QRS–ST–T complex is recognized in leads II and aVF and possibly in lead III in association with concomitant reciprocal or mirror image in leads V1–V5. The ST-segment deviation vector is directed leftwards and more posteriorly than downwards suggesting a culprit lesion proximal to an obtuse marginal branch of a not so dominant LCx artery (J wave in lead aVL that is directionally opposite to the inferior wall and faces the high anterior-lateral wall in the base of the left ventricle) also supplying a large posterior LV branch causing transmural ischaemia of the inferior-lateral LV wall toward the apex (negative J wave in lead aVR). Indeed, the anatomy of the culprit artery and the location of the culprit lesion as presented in the angiographic image illustrating the article in question fit nicely with the above-mentioned anatomical information extracted from the ECG. Accordingly, this case corroborates and simultaneously highlights the utility of ischaemia-induced J waves in localizing the infarct-related artery. Furthermore, recognition of J wave dynamics during myocardial ischaemia (J wave augmentation and merging with the ST-segment forming a lambda-like ST-segment elevation pattern) is important because it denotes an increased risk of Phase II reentry and subsequent VF, thereby facilitating choosing the most effective therapeutic approaches. A genetically determined electrophysiological milieu may be present in those patients who manifest prominent J waves during acute myocardial ischaemia whether or not they develop VF. Consequently, such patients face a high life-long risk of sudden cardiac death during recurrent ischaemia, something that should be considered when placement of an intracardiac defibrillator is contemplated.

Funding

The author received no financial support for the research, authorship, and/or publication of this article.

References

1. Sato A, Tanabe Y, Chinushi M, Hayashi Y, Yoshida T, Ito E, Izumi D, Iijima K, Yagihara N, Watanabe H, Furushima H, Aizawa Y. Analysis of J waves during myocardial ischaemia. *Europace* 2012;**14**:715–723. doi:10.1093/europace/eur323
2. Andreou AY. Acute coronary syndrome featuring dynamic J waves. *Cardiol J* 2021;**28**:638–639. doi:10.5603/CJ.2021.0069
3. Andreou AY. Cardiac arrest in a patient with acute ST-segment elevation myocardial infarction. *Tex Heart Inst J*. Forthcoming. doi:10.14503/THIJ-21-7792
4. Yan GX, Antzelevitch C. Cellular basis for the electrocardiographic J wave. *Circulation* 1996;**93**:372–379. doi:10.1161/01.CIR.93.2.372
5. Cha KC, Ahn SG, Hwang SO. Ischaemia-induced Osborn waves. *Eur Heart J Case Rep* 2022;**6**:ytac143. doi:10.1093/ehjcr/ytac143

Andreas Yiangos Andreou[1,2]*
[1]Department of Cardiology, Limassol General Hospital, Nikeas Street, Pano Polemidia, Postal code 3304, PO Box 56060, Limassol, Cyprus; [2]University of Nicosia Medical School, Nicosia, Cyprus

* Corresponding author. Tel: +357 25801437, Fax: +357 25801432, Email: y.andreas@yahoo.com

Handling Editor: Christian Fielder Camm