Central apneas and Ticagrelor related dyspnea in patients with acute coronary syndrome

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Funding Acknowledgement: Type of funding source: None

Background: Patients treated with ticagrelor often develop dyspnea of unknown origin. We aim to explore the contribution of central apneas to ticagrelor-related dyspnea in patients with acute coronary syndrome (ACS).

Methods: We consecutively enrolled patients with ACS, preserved left ventricular ejection fraction and no history of obstructive sleep apnea, treated either with ticagrelor 90 mg bid (n=30) or prasugrel 10 mg od (n=24). One week after ACS onset, all patients underwent, beyond thorough cardiovascular and respiratory assessment, 24-hour cardiorespiratory monitoring and assessment of chemosensitivity to hypercapnia.

Results: Patients treated with ticagrelor reported more frequently dyspnea than patients treated with prasugrel (43% versus 4%, p=0.001), despite no difference in demographic, clinical, echocardiographic and pulmonary data. Patients with dyspnea induced by ticagrelor showed higher apnea-hypopnea and central apnea index both at daytime and at nighttime compared to patients treated with ticagrelor but without dyspnea and patients treated with prasugrel (daytime AHI: 26 [7–34] vs 6 [4–14] and 6 [0–11] events/hour; nighttime AHI: 65 [17–72] vs 22 [8–37] and vs 11 [4–23] events/hour; daytime CAI: 5 [1–15] vs 1 [0–6] and 0 [0–1) events/hour; nighttime CAI 34 [2–55] vs 3 [0–9] and 0 [0–1], all $p<0.05$). Likewise, they also presented with higher hypercapnic ventilatory response (2.4 [1.9–2.7] vs 1.3 [1.1–1.9] and 0.9 [0.5–2.1] L/min/mmHg, all $p<0.05$).

Conclusions: Central apneas should be considered a likely mechanism of dyspnea in ACS patients treated with ticagrelor. A drug-related sensitization of the chemoreflex may be the cause of ventilatory instability in this setting.