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Comment on "Delayed-Onset Type 1 Kounis Syndrome Caused Ventricular Fibrillation: A Case Report"

LETTER TO THE EDITOR

To the Editor,

We read with interest the case by Cui et al. describing delayed-onset Type 1 Kounis syndrome triggered by levofloxacin and complicated by ventricular fibrillation (VF).[1] The report highlights the overlap between allergic activation and coronary vasoreactivity but raises several diagnostic and therapeutic concerns.

In this case, aspirin 300 mg and clopidogrel 300 mg were given before angiography despite normal cardiac biomarkers. The 2023 European Society of Cardiology guidelines advise against pretreatment with $P2Y_{12}$ inhibitors when an early invasive strategy is planned, recommending loading only after coronary anatomy is known.[2] Initial management of allergic coronary syndromes may focus on anti-allergic and vasodilator therapy. Observational evidence also discourages dual antiplatelet therapy (DAPT) in vasospastic settings. In the VA-Korea Registry of 1,838 patients with vasospastic angina, DAPT increased adverse outcomes compared with no antiplatelet therapy, whereas aspirin alone was neutral.[3]

A further issue is the unreported QTc interval. Fluoroquinolones, including levofloxacin, can prolong repolarization and provoke torsades de pointes or VF, especially with ischemia or electrolyte imbalance.[4] The electrocardiogram in this case suggests a QTc near 480 ms, yet QT or electrolyte data were absent. Levofloxacin-induced QT prolongation could thus have contributed to arrhythmogenesis. Serial QTc and electrolyte monitoring would help determine whether the arrhythmia was resulted from vasospasm, repolarization delay, or both.

Finally, vasospastic angina was presumed but not confirmed because no ergonovine provocation test was performed after stabilization. Such testing remains the diagnostic standard for coronary spasm in patients with non-obstructive angiography and compatible symptoms, improving diagnostic certainty and guiding treatment.[5]

In summary, early DAPT may worsen outcomes in allergic coronary syndromes, QT-active antibiotics can independently precipitate malignant arrhythmia, and provocation testing is essential for accurate diagnosis. Prompt anti-allergic and vasodilator therapy, QT monitoring, and anatomy-guided antiplatelet selection represent safer, evidence-based management.

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