

Kounis syndrome, POTS, arrhythmias:

the heart under mast cell influence

Why the heart?

The heart and blood vessels are among the most sensitive targets of mast cell activation. Cardiac mast cells — present in high density in peri-vascular tissue, the pericardium and the valves — release, upon degranulation, mediators with direct cardiovascular action: histamine (vasodilation, bradycardia or tachycardia), tryptase (coagulation activation), leukotrienes (vasospasm), prostaglandins (arrhythmogenic). In a PMCHS terrain, this activation is chronic and low-grade — producing cardiac presentations often labelled 'functional' or 'anxiety-related' for lack of an identified mechanism.

Kounis Syndrome

Kounis syndrome is the occurrence of an acute coronary syndrome (angina, myocardial infarction) in the context of mast cell activation — allergy, drug intolerance, insect sting, food trigger. It is underdiagnosed because the link between the triggering event and the cardiac symptomatology is not systematically investigated. In the PMCHS terrain, the activation threshold is lowered: minor stimuli are sufficient to provoke mast cell-driven coronary spasm.

“ Any chest pain occurring within minutes of a food, drug or allergic exposure should raise suspicion of Kounis syndrome in a PMCHS profile. ”

POTS and mast cell dysautonomia

POTS (postural orthostatic tachycardia syndrome) is frequently associated with MCAS and the PMCHS terrain. Mechanism: mast cell degranulation induces excessive peripheral vasodilation, to which the autonomic nervous system responds with compensatory tachycardia. Orthostatic intolerance, dizziness on standing, postural fatigue and post-exertional brain fog are explained by this mast cell-driven vasomotor instability.

The link with vagal dysautonomia is particularly relevant in the PMCHS framework: mast cell tryptase degrades the myelin of autonomic nerve fibres, reducing vagal tone and facilitating reflex tachycardia episodes.

Arrhythmias and unexplained palpitations

Palpitations, ectopic beats and paroxysmal tachycardia without identifiable structural cardiopathy are frequent in PMCHS profiles. Histamine acts directly on cardiac ion channels (H1 and H2 channels on nodal cells), potentially triggering rhythm disorders during degranulation peaks. These episodes are often correlated with meals, stress, hormonal cycles or seasonal changes — all mast cell degranulation factors.

What this means in practice

- **Negative cardiac workup** ≠ **no cause**. Standard investigations do not explore mast cell origin.
- Keep a **symptom diary**: time, preceding meal, stress, exposure. Correlation with triggers is diagnostic.
- **Mast cell stabilisation** (quercetin, luteolin, H1+H2 antihistamines) can significantly reduce the frequency of rhythm episodes and orthostatic episodes.
- If Kounis syndrome is suspected, **inform your cardiologist** of the PMCHS/MCAS terrain — acute management differs.