

## Does pheochromocytoma mimic or cause acute myocardial infarction?

In their instructive paper, Musuraca et al. [1] remind us of the possibility that myocardial damage with the clinical picture of acute coronary syndrome (ACS) may result from pheochromocytoma (Pheo). We strongly agree that keeping in mind and searching for atypical causes of ACS may ‘help avoid mistakes in their treatment’ [1, 2].

On the other hand, we think that it is worth discussing whether Pheo in this patient *mimics* ACS or *causes* ACS. Namely, according to the universal definition of myocardial infarction (MI): the term MI reflects cell death of cardiac myocytes caused by ischemia, which is the result of a perfusion imbalance between supply and demand [3]. Ischemia in a clinical setting can most often be identified from the patient’s *history* and from the *ECG* [3].

The patient had a typical ACS presentation (chest pain), indicating an ischemic event. The akinesia of the septum and anterolateral wall (regional abnormality) is also suggestive of ischemic cause. The other required item for a diagnosis of MI, i.e. myocardial necrosis, was also present (elevated troponin). Thus, the patient had *both conditions required for acute MI*: (1) ischemia, as confirmed by a positive history of chest pain and additionally suggested by regional echocardiographic abnormality of contraction, and (2) myocardial necrosis, where the troponin concentration was increased. Moreover, conditions which are well known to cause ischemia were present (high blood pressure and tachycardia), adding proofs of typical clinical milieu. Accordingly, it was apparent that this patient had acute MI (AMI). Type 1 or Type 2? It is impossible to answer, because no coronary angiography result is reported. *Type 2* MI occurs secondary to ischemia due to either increased oxygen demand or decreased supply, e.g. coronary artery spasm, coronary embolism, anaemia, arrhythmias, hypertension, or hypotension [3].

In this patient, one cannot confirm ‘primary’ ischemia, but there were some of the main causes of the ‘secondary’ ischemia: (1) increased myocardial oxygen consumption (heart rate was up to 170 beats per minute and systolic blood pressure was

up to 220 mm Hg, with likely positive inotropic effect of catecholamines) as well as (2) decreased oxygen supply (short diastole due to rapid heart rate which produced reduction in coronary blood flow, occurring mostly in diastole; also drop of systolic blood pressure to 70 mm Hg may compromise oxygen supply). All the above-mentioned mechanisms suggest Type 2 MI. Was there also a coronary artery spasm operating? We cannot know this, but in hyperadrenergic states, spasm has been a well-known causative mechanism of AMI.

Additionally, it has been shown that coronary artery spasm may produce thrombosis. This is logical, as stasis induces thrombosis (as has been known since R. Virchow).

In conclusion, Pheo may mimic, but may also cause AMI (which is what we believe happened in the patient reported). Moreover, doctors should be aware:

- of the possibility of underlining Pheo in each patient with ACS;
- of the many cardiovascular diseases that may be caused by Pheo: arterial hypertension (AHT), tachycardia, arrhythmia, ventricular hypertrophy, dilated cardiomyopathy, congestive heart failure, transient left ventricular apical ballooning, acute MI, aortic dissection, etc [4];
- that although AHT (especially if severe, with crises and with paradoxical orthostatic hypotension) is suggestive of Pheo, AHT is not always present; Pheo with predominant epinephrine or dopamine secretion may take a hypotensive course [5]. Moreover, the patient may present in shock.

### References

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