

Letters to the editor

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The tako-tsubo syndrome and bronchial asthma: the chicken or the egg dilemma

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The tako-tsubo syndrome is considered very popular among modern cardiologists in consideration of the utmost confusing clinical presentation whereas the coronary anatomy is, hopefully, normal.

A 72-year-old male with a history of bronchial asthma was admitted to our coronary care unit after an acute asthmatic attack which the patient experienced alone in his apartment and which frightened him very much, as stated on admission. Peculiarly, his ECG showed ST segment elevation in anterior leads and ultrasound features were typical of apical ballooning together with a depressed 37% ejection fraction.

Actually, the confusing physical finding was dyspnoea in the absence of any kind of chest pain or typical pain in other regions that could have led to a high suspicion of coronary artery disease (CAD).

On the same day the patient underwent coronary angiography which was normal – no big surprise to all staff cardiologists, even in consideration of the dire clinical presentation and of the only fourfold increase of TnI.

On day two, ST segment elevation resolved and giant negative T waves appeared in precordial leads together with QT prolongation, which is a fairly common feature of tako-tsubo syndrome.

On day six the patient was discharged uneventfully, with a normal ECG and cardiac ultrasound examination and, best of all, no more wheezing.

To our knowledge this is the second description of a case of tako-tsubo syndrome following an acute asthmatic attack [1]. We thought it could be useful to keep in mind that any kind of psychological stress may ‘frighten’ the heart as well during the catecholamine storm of the ancestral fight-or-flight response. Unfortunately, in most cases we have very few hints in order to rule out CAD, in order to avoid any

specific and potentially harmful treatment. As to pulmonary disease, Hertting *et al.* [2] found that 44% of their patient population actually had chronic obstructive pulmonary disease or asthma, but it is still difficult to know whether asthma itself triggers the syndrome or is only associated with a higher risk of apical ballooning, as a reinforcement of the chicken or the egg causality dilemma.

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Beware of, not only the dogs, but the passionate kissing and the Kounis syndrome

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Kounis syndrome [1] is the association of acute coronary syndromes with conditions involving mast cell activation and interrelated and interacting macrophages and T cells, including allergic or hypersensitivity and anaphylactic or anaphylactoid insults. It is caused by inflammatory mediators such as histamine, neutral proteases, arachidonic acid products, platelet activating factor and a variety of cytokines and chemokines released during the activation process. Platelets with high (FcεRI) and/or low (FcγRII) affinity IgE receptors in their surface could be activated by corresponding antigens and contribute to the thrombotic process [2].

Atopic diseases and conditions such as food allergy, drugs such as antibiotics, environmental exposures such as stings and contacting allergens are some of its causes which currently are associated with stent thrombosis [3].

In the very interesting study published in this journal [4] an atopic patient, sensitized to beta-lactams, with two previous myocardial infarctions following amoxicillin oral intake, developed, for a third time, an acute myocardial infarction after he was licked on the face by his dedicated dog. These events constitute characteristic clinical manifestation of the so-called allergic myocardial infarction or Kounis syndrome. Beta-lactams are some of the usual causes of Kounis syndrome and in a recent study 17 patients were confirmed, during cardiac catheterization, to have developed the type I and the type II variants of this syndrome [5].

A sensitized individual does not always need to ingest the culpable allergen to develop this syndrome. In a survey of people who experienced allergic reactions to peanuts served on commercial airlines, 48% had eaten the peanuts, 33% had inhaled peanut allergen and 19% had skin contact [6]. In another survey of 379 people with self-reported histories compatible with IgE-mediated food allergy, 20 (5.3%) reported reactions from kissing [7]. Therefore, allergens seem to be conveyed from one person to the other not only by contact or ingesting but by inhaling, touching, eating, smelling, licking or kissing.

Kissing or licking-induced allergic reactions with cardiovascular involvement may be a very dangerous condition because such a reaction is probably far from the mind of most of people with atopy. The 'kiss of death' has already been described [8] as clinical reality.

In the described case the dog had been treated with amoxicillin, and a salivary concentration of amoxicillin is

sufficient to induce allergic reaction and Kounis syndrome when such a saliva comes in touch with the skin of an atopic patient. Sputum concentrations of amoxicillin are at the level of 0.4–0.5 mg/l after 750 mg amoxicillin oral administration [9].

Therefore, atopic individuals sensitized to drugs should always bear in mind that these allergens can be conveyed via several routes and should comply with the paraphrased version of the ancient latin dictum: 'timeo dogs et dona ferentes'.

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