

current airway guidelines [1-4] recommend management of the CICV situation using cannula cricothyroidotomy with percutaneous transtracheal jet ventilation or surgical cricothyroidotomy. Most studies comparing the efficacy of different emergency airway equipment in simulated patients, cadavers, and animal models did not evaluate the different devices with regard to adequate ventilation. The available studies suggest that initially all systems might be able to inflate the lungs to 0.5 L, but that ventilation then fails rapidly within 60 seconds if needle cricothyroidotomy is used with a low-pressure ($15 \text{ l} \times \text{min}^{-1}$) ventilation system [5]. Evidence from model lung studies shows that a needle airway failed to provide good ventilation in the presence of upper airway obstruction because of inadequate exhalation via the narrow cannula lumen [6]. Dworkin et al [7] found that regardless of jet ventilator settings and lung parameters, exhalation time becomes rapidly prolonged as the upper airway is restricted beyond the critical diameter of 4.0 to 4.5 mm, after which air trapping occurs. A dog model study by Ward et al [8] suggests, however, that needle cricothyroidotomy can provide effective ventilation in the presence of increasing airway obstruction, provided it is used in conjunction with a jet ventilator. It should be noted that jet ventilators are rarely, if ever, available in the prehospital setting. The lung model data also show that in the presence of upper airway obstruction, both low-pressure and high-pressure jet ventilation via a cannula may cause dangerously high airway pressures capable of causing barotrauma [9-12]. In contrast, a surgical airway provides effective ventilation at lower pressures regardless of the degree of restriction of the upper airway [6].

Because most prehospital cricothyroidotomies are performed to manage immediately life-threatening injuries and illnesses involving obstruction of the upper airway, the practical clinical significance of these findings is that needle cricothyroidotomy is only advocated 'if an appropriate ventilator is immediately available' [3].

Moreover, in a patient with a short, fat neck, blind insertion of a transtracheal catheter may not prove to be the safer and simpler technique.

We believe that in this particular case, the early abandonment of alternative procedures, which would have most likely failed in the patient described anyway, proved lifesaving.

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Hypersensitivity myocarditis and hypersensitivity coronary syndrome (Kounis syndrome)

To the Editor,

There are 2 cardiac diseases of hypersensitivity etiology that affect the myocardium and the coronary arteries, respectively. The first is an inflammatory disease affecting the myocardium and the cardiac conduction system manifesting as a complication of drug therapy [1]. The second is the concurrence of acute coronary syndromes with conditions associated with mast cell activation, involving inter-related and interacting inflammatory cells, and including allergic or hypersensitivity and anaphylactic or anaphylactoid insults [2].

In the very interesting report published in this journal [3], a young man 23 years of age developed skin eruptions in hands, lips, and mouth, and ulcerated lesion in the penis after taking 2 pills of flurbiprofen because of headache 10 days ago. Contemporarily, he had cardiac involvement with ST elevation, increased troponin I, increased cardiac enzymes, and inferoseptal myocardial ischemia at rest resolving at redistribution in TI-201 rest-redistribution myocardial perfusion single photon emission computed tomography.

The authors of this report suspected that the patient's condition was a drug-induced myocarditis. However, they omitted to refer to Kounis syndrome, which shares nearly the same symptoms and laboratory findings with hypersensitivity myocarditis (Table 1). The only differences between these 2 hypersensitivity conditions are the presence of eosinophils, atypical lymphocytes, and giant cells in myocardial biopsy in hypersensitivity myocarditis; whereas biopsy in Kounis

syndrome is typically normal. The normal coronary angiogram in hypersensitivity myocarditis; whereas angiogram in Kounis syndrome, especially in type II variant [4], shows coronary artery disease. Furthermore, mildly elevated cardiac enzymes and mild chest pain are characteristics of drug-induced myocarditis while eosinophilia may be absent in both conditions. In this patient, coronary angiography was not performed, although single photon emission computed tomography revealed some inferoseptal ischemia at rest with significantly raised cardiac enzymes and troponin I and myocardial biopsy was not attempted because of the risk of complications. The latter, as the authors correctly emphasized, is required to establish a definite diagnosis.

Hypersensitivity myocarditis can affect individuals of any age, and cases of Kounis syndrome have been reported in juveniles [5]. Ideally, in this patient, allergic screening with measurement of mast cell mediators such as histamine, tryptase, chymase, and arachidonic acid metabolites should have been carried out to exclude contemporary coronary mast cell activation.

The treatment of both conditions is the same including corticosteroids, H1 and H2 blockers; and in severe and persistent cases, the addition of leukotriene antagonists and immunosuppressives may be life saving. However, this report shows that hypersensitivity myocarditis and Kounis syndrome, although not rare diseases, are very rarely diagnosed and differentiated diseases!

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Table 1 Cardiac features of suspected hypersensitivity myocarditis and hypersensitivity coronary syndrome (Kounis syndrome)

	Hypersensitivity myocarditis	Kounis syndrome
Cardiac symptoms		
Acute chest pain	–	+
Chest discomfort	+	+
Dyspnea	+	+
Palpitations	+	+
Sudden death	+	+
Cardiac signs		
Elevated JVP	+	+
Irregular pulse	+	+
Gallop rhythm	+	+
Electrocardiographic signs		
Atrioventricular block	+	+
Left bundle-branch block	+	+
Right bundle-branch block	+	+
Sinus tachycardia	+	+
ST-segment elevation	+	+
ST-segment depression	+	+
T-wave inversion	+	+
Ventricular tachycardia	+	+
Laboratory signs		
Coronary angiography	+	±
Eosinophilia	–	±
Increased cardiac enzymes and especially CPK-MB	+	+
Increased troponins	+	+
Cardiomegaly in the chest x-ray	+	+
Dilated cardiac chambers in echogram	+	+
Eosinophils, atypical lymphocytes, and giant cells in biopsy	±	–

JVP indicates jugular vein pressure; CPK-MB, creatinine phosphokinase-MB.

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Is it Kounis syndrome or myocarditis?

To the Editor,

We have read "Kounis syndrome" and surprised to see the common points of this disease with "hypersensitivity myocarditis," which we reported a case recently [1]. We believe that most of the emergency physicians have knowledge about drug-induced acute coronary syndrome, bee stings or viper venom poisoning-related myocardial infarction, but only a few of us know that all these coronary syndromes come out to a common pathophysiologic mechanism and a common name: hypersensitivity coronary syndrome (Kounis syndrome) [2]. Thus, we believe that this correspondence will aid and educate emergency physicians and will also reduce misdiagnosis and wrong treatment. However, there are some problems about the content of this article and the examples that are mentioned in the article.

The article offers that our case was a Kounis syndrome rather than a "hypersensitivity myocarditis." However, the case has contradictions about that diagnosis in the following points:

1. To our knowledge, Kounis syndrome appears in the first 24 hours after the event occurs. However, in our case, the tableau appeared more than 1 week later.
2. In Kounis syndrome, the electrocardiogram generally shows local ST elevation, which is compatible with infarction (eg, inferior or anterior wall of the heart). On the contrary, in our case, the electrocardiogram showed an ST elevation of 0.5 to 1 mV in all derivations.
3. The chest pain in Kounis syndrome is a typical ischemic angina pectoris. Although the chest pain is a subjective criteria, in our case, it was compatible with nonischemic chest pain.
4. According to the author, Kounis syndrome was well defined and well typed before in his articles [1]: "Chest pain, during an allergic insult, with electrocardiographic ischemic changes but with normal cardiac

enzymes, negative toponins, normal sestamibi scan, normal coronary angiogram and positive ergonovine or histamine test are in favour of type I variant of the syndrome. Acute allergic reactions associated with acute myocardial infarction with angiographic evidence of coronary artery disease constitute type II variant of Kounis syndrome." However, this well-defined situation conflicts with this correspondence. As you know, in our case, the myocardial perfusion scintigraphy was positive; the biomarkers (including troponin I) were elevated. If we take into account that our patient was a 23-year-old man and have no coronary risk factor, it is hard to classify our patient to any type of Kounis syndrome. Also, the table in the correspondence is incompetent.

5. Myocardial perfusion scintigraphy results of our patient were not normal and revealed a resolved defect in the inferoseptal wall. According to Kounis, "acute allergic reactions associated with acute myocardial infarction with angiographic evidence of coronary artery disease constitute type II variant of Kounis syndrome". In our case, scintigraphic results (resolved defect in the inferoseptal wall) did not support the myocardial infarction [2-4].

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