

Troponin T Should Not Be Considered as a Screening Test for Pediatric Myocarditis

To the Editors:

We greatly appreciated the article by Chong Shu-Ling and colleagues about pediatric myocarditis. In the discussion, the authors seem to suggest that troponin T may be a useful test for identifying myocarditis in children.

We take exception to this possible conclusion.

Cardiac troponins I and T are contractile proteins unique to cardiac muscle cells and are released into the blood within hours following cardiac muscle cell injury or death, even though isolated troponin increases are not specific enough to identify children affected by myocarditis.

Renko et al¹ screened 1009 children with upper respiratory infections. Troponin I was above the limit in 8 cases: 6 of them (5 were <30 days old) had respiratory infections; 1 patient—aged 15—had a pharyngitis, and another boy was hospitalized on account of symptomatic perimyocarditis.

Brown and colleagues² studied 212 patients younger than 22 years admitted to the emergency department for chest pain: troponin levels were increased in 37 subjects (17%). Ten patients had a diagnosis of myocarditis or pericarditis, whereas 27 had no myocardial involvement. Both authors stated that troponin increase is not specific for myocarditis.

In the last 4 years (2009–2012), in the emergency department of our third-level children's hospital, high troponin levels were detected in 18 patients with a suspicion of myocardial disease, but only 2 of them had a diagnosis of myocarditis or pericarditis.

Troponin levels were detected in 131 patients aged 7.1 ± 6.4 years old. Upon the whole, 18 troponin levels (13.7%) were positive (cutoff >0.01 ng/L), and only 2 of them (11%) had a diagnosis of cardiac disease: myocarditis and perimyocarditis whose cardioselective markers resulted considerably increased (>120 ng/L).

The other patients were discharged with different diagnoses: 11 (61%) respiratory diseases (pneumothorax, bronchiolitis, pneumonias, viral infections), 2 intoxications (11%), 2 vasovagal syncope (11%), and 1 cyclic vomiting syndrome.

Available evidence^{1,2} suggests that troponin is a sensible but not a specific marker, and its increase is not specific enough to consider appropriated decision to test it in the emergency department, to identify children affected by myocarditis, especially in younger children with respiratory infections. A false-positive test can lead to unuseful and repeated investigation. We believe that troponin should be performed only in signs suggestive of cardiopathy after an electrocardiogram and chest radiograph.

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DISCLOSURE

The authors declare no conflict of interest.

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Dealing With Ketamine Sedation Adverse Events: Are Coadministered Anticholinergics Necessary?

To the Editors:

It was with great interest that we read the clinical trial "Ketamine and Atropine for Pediatric Sedation—A Prospective Double-blind Randomized Controlled Trial," published in the February issue of *Pediatric Emergency Care* by Dr Asadi and colleagues.¹ They concluded that the incidence of hypersalivation was statistically

lower in the pretreated group with atropine, if compared with the control group, which received distilled water as placebo (12% vs 28%; odds ratio, 0.37; [95% confidence interval, 0.1852–0.738]).

Ketamine produces potent analgesia, sedation, and amnesia, with some characteristics that require separate consideration from other sedative agents. Its dissociative sedation is obtained by disconnection between the limbic and thalamocortical systems, isolating the child from outside sensorial and painful stimuli.² Despite having been used for more than 20 years for procedural sedation in children, adverse events with ketamine sedation are still a challenge in the pediatric emergency department scenario. Thus, many alternatives have been studied to prevent or minimize these events.

The largest meta-analysis about adverse events associated with ketamine was published in 2009 by Dr Green and colleagues.³ In 8282 pediatric ketamine sedations, performed in 32 emergency departments, some of the most important independent predictors of airway and respiratory adverse events were age (<2 and ≥ 13 years old), high intravenous dose (>2.5 mg/kg), and coadministered anticholinergics (odds ratio, 1.82; 95% confidence interval, 1.36–2.42). In this study, the authors state that this association was unexpected, and because of age being a potential confounding variable in this association, an adjusted analysis for age confirmed those findings. Later, in 2011, an update of a clinical practice guideline for ketamine dissociative sedation did not support the routinely prescription of anticholinergics to reduce secretions; however, this approach could be reserved for children with important hypersalivation or an impaired ability to mobilize secretions.⁴

The prophylactic coadministration of an anticholinergic (atropine or glycopyrrolate) is usually recommended, with the intent of reducing oral secretions and potential airway adverse events.⁵ The study by Dr Asadi et al¹ has a sound methodology, with a great advantage on its design (clinical trial), with an adequate sample size and clear outcome parameters. However, authors were not clear about the clinical magnitude of their findings, as most children with hypersalivation are not at higher risks for airway adverse events. In addition to these conclusions, the efficacy of ketamine sedation does not

seem to be influenced by anticholinergics premedication. Overall, the incidence of adverse events was low in both groups (control and intervention), and statistical analysis showed that these events were probably not influenced by the administration of atropine before sedation.

In conclusion, evidence for routinely prescription of anticholinergics is low in the ketamine sedation setting. Nevertheless, it can lead to higher incidences of airway respiratory events. Patients should be carefully selected to receive atropine before dissociative sedation.

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