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Correspondence

Epinephrine-induced myocardial infarction in severe anaphylaxis: is β -blocker a bad actor or bystander? ☆☆☆

To the Editor,

It was interesting to review the recent case report by Cunningham et al [1]. The authors had linked the prior use of nonselective β -blocker as a predisposing factor for the myocardial ischemia that occurred after epinephrine administration. Although no epidemiological studies point out that anaphylaxis occurs more frequently in patients taking β -blockers [2], it is more likely that, in this patient, coronary vasoconstriction would have occurred due to repeated boluses of epinephrine.

Interestingly, Alam et al [3] from the Cleveland clinic have reported that the fatality rate of anaphylaxis patients on β -blockers and those who were not was 3%, and 8.5%, respectively. They also found no significant difference between the 2 groups for surviving resuscitation, length of hospital stay, and 1-month survival. Another point of curiosity is that was there any hard evidence of myocardial necrosis or dysfunction because it is well known that elevated troponin could be due to a myriad of etiological factors [4]. The decision to withhold or discontinue β -blockers must be considered carefully from the perspective of risk vs benefit for each individual. Furthermore, in this case, advice of an autoinjector needs to be relooked at because this patient developed drug-induced anaphylaxis and because the authors incriminate epinephrine to be the culprit for myocardial ischemia.

Therapeutically, intravenous injection of glucagon may be beneficial in these cases, as glucagon has inotropic, chronotropic, and vasoactive properties independent of β -receptors. In addition, it stimulates endogenous catecholamine release by activating adenylyl cyclase. Thus, glucagon overcomes myocardial ischemia as well as overrides the paradoxical response to epinephrine [5].

From the point of education and training, all those involved in the treatment of anaphylaxis shall be taught and trained to consider adverse effects, nonresponsiveness and paradoxical response of epinephrine, and the alternatives under such circumstances.

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