Author's reply

Sir,
We thank the authors for their interest and their valuable comments on our article.

Though, their views and correlation between anaphylactic/allergic mechanisms with the impending cardiac events and coronary disease are exciting and interesting, a close correlation between the two has already been suggested ages ago and that the novel syndrome depicts no new features. The main cardiovascular events during anaphylaxis are a result of fluid extravasation and vasodilation as a result of histamine release, causing a mixed distributive-hypovolaemic type of shock that ultimately, results in cardiac decompensation and myocardial ischemia.[1]

Further, coronary artery spasm can occur and can lead to subsequent myocardial infarction, dysrhythmia, or cardiac arrest as a result of generalized massive release of histamine from the sensitized mast cells. Those with underlying coronary disease are at greater risk of cardiac effects from such drug induced anaphylaxis. Though, profound hypotension and tachycardia has been used clinically to differentiate an anaphylactic event from a vasovagal reaction,[2] a cardioinhibitory Bezold-Jarisch reflex has also been described in 10% of cases, where a slow heart rate is also associated with hypotension.[3]

So the correlation, extrapolation and support of evidence of the authors, to this case states that, cardiac damage is the primary event during such allergic/anaphylactic reactions remains a doubtful issue suggesting that, the human heart cannot be a primary site in anaphylaxis. Further, the stated results from the animal models of anaphylaxis, the released mediators, and tissue targets do not reflect the same as in humans.[4] Besides there is hardly any difference between anaphylaxis and the described syndrome.

Diagnosis of invasive pulmonary aspergillosis was done in our patient on the basis of isolation of an Aspergillus species from the sputum and from bronchoalveolar lavage samples. Pericardial and myocardial aspergillosis are though rare manifestations of systemic aspergillosis, cardiac involvement by aspergillus species, may result from the contagious spread from the lungs or by the hematogenous route of spread which can also result in arrhythmias and death.[5] Hence, the myocardial ischemic signs and symptoms of our patient might be either due to allergic or hypersensitivity and anaphylactic or anaphylactoid reactions due to the drug or might be a result of disseminated systemic aspergillosis.

Though, serum tryptase and urinary N-methylhistamine levels are useful markers in confirming the diagnosis of anaphylaxis, these tests must be obtained in close proximity to the anaphylactic reaction to be useful. The reasons being that the serum tryptase levels as well as the urinary N-methylhistamine levels peaks at 1 h with a serum half-life of approximately 2 h. On the other hand, there is an evidence of massive elevated levels of serum tryptase (>50U/L) in cases of fatal anaphylaxis, when measured during post mortem examinations suggesting it to be a specific indicator of the severity of anaphylaxis. But in our case, since permission for postmortem was not provided by the family hence the levels could not be measured.[6]

However, it is very true to state that, echinocadins such as caspofungin, a basic polypeptide that is the semi-synthetic derivative of pneumocandin B0, a fermentation product of *Glarea lozoyensis*. can act as antigens leading to anaphylaxis as well as serious hypersensitivity reactions.

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