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Letter to the Editor

Determination of specific IgE in pericardial fluid

Dear Sir,

Koniari et al. [1] have recently commented on an article published in *Forensic Science International* [2], which focused on the determination of total and specific IgE in the pericardial and cerebrospinal fluid for diagnostic purposes in the forensic setting.

Koniari et al. [1] properly highlighted that the heart may be the primary target of anaphylaxis, especially regarding the coronary arteries. According to this line of reasoning, pericardial fluid might potentially provide important data in some cases of sudden unexpected death of cardiac origin.

The composition of normal human pericardial fluid has been difficult to define since all available data have been obtained through cardiothoracic surgery patients or animals, likely compromising its validity, though pericardial fluid is considered to be a plasma ultrafiltration product, as other serosal cavity fluids. Studies in greyhounds showed that the concentrations of sodium, chloride, calcium and magnesium were lower in pericardial fluid than in plasma. On the contrary, the concentration of potassium was higher than in plasma, attributed to potassium leakage from the myocardial interstitium toward the pericardial cavity during systole [3].

In the realm of forensic pathology, several specific molecules have been measured in pericardial fluid for diagnostic purposes, including some related to anaphylaxis (mast cell tryptase) and myocardial function or damage (troponin I, troponin T, creatine kinase, myoglobin and natriuretic peptides) [4–10].

For instance, an interesting observation was made regarding individuals who had undergone postmortem investigations within 24 h after death following contrast material administration. Increased levels of mast cell tryptase in postmortem serum from femoral blood were observed accompanied by increased levels of mast cell tryptase in pericardial fluid. However, mast cell tryptase concentrations in corpses with causes of death unrelated to anaphylaxis and severe decomposition changes were also characterized by extremely high levels of mast cell tryptase in pericardial fluid, likely suggesting that postmortem changes might be associated with massive mast cell tryptase leakage from the arterial vessel wall into the pericardial space [4].

This finding is not surprising since mast cells are inflammatory cells that accumulate in the coronary artery wall at the actual sites of atherosclerotic plaque erosion or rupture, where their number has been found up to 200-fold higher than in unaffected areas of the vessel wall. Moreover, an increased number of activated mast cells in the coronary wall (intima, media and adventitia) has been

noted in individuals who died from coronary artery spasm attacks [1,11].

Like pericardial fluid tryptase, six to ten times higher levels of cardiac troponin T and I in pericardial fluid compared to postmortem serum values have been postulated to be caused by the proximity of the myocardium to the pericardial space, with the possible direct leakage of these molecules into pericardial space. This has led some authors to conclude that pericardial troponin I measurement to determine the extent of myocardial injury (using a micro-particle enzyme immunoassay technique) was not as useful as the assay in blood, whereas other authors found that cardiac troponin T determination in pericardial fluid by a high sensitive technique could be considered a reliable diagnostic test in the postmortem diagnosis of sudden cardiac death, with sensitivity and specificity values close to those found in *in vivo* samples [5,6].

Analogously, some researchers have suggested that the time after death should be taken into consideration when cardiac troponins are measured in postmortem samples by conventional assays. Some researchers have asserted that postmortem levels can change depending on cause of death and postmortem interval length, while others have failed to find significant correlations between postmortem serum/pericardial fluid troponin concentrations and autolysis time [5–10].

To conclude, we fully support the observations of Koniari et al. [1], who emphasized the importance of investigating various tissues that might have been infiltrated by allergic inflammatory cells, including the pericardium, in order to identify potential cases of sudden anaphylactic deaths.

On the other hand, the interpretation of biochemical results obtained from laboratory analyses performed in pericardial fluid collected at autopsy could result in a challenging issue due to the difficultly definable role of a plethora of factors. These include myocardium/vascular wall to pericardial space proximity, possible direct leakage of some molecules from the epicardium (or the coronary artery wall) into the pericardial fluid, postmortem interval, decomposition changes, and chosen analytical technique for a specific analyte, which may all play a more or less significant role in modifying pericardial fluid composition after death and therefore affect the laboratory results obtained.

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