H127 The Loss of Dystrophin: An Immunohistochemical Study for Postmortem Diagnosis of Early Myocardial Ischemia

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After attending this presentation, attendees will understand the importance of being informed regarding the detection of immuno-inflammatory and cellular phenomena accompanying cardiac alterations during the early inflammatory phase to aid in the forensic diagnosis of early ischemic damage.

This presentation will impact the forensic science community by providing a new tool to detect early myocardial ischemic damage through the immunohistochemical analysis of dystrophin.

Sudden Cardiac Death (SCD) is an unexpected natural death due to cardiac causes that occurs within a short time period in a person without any prior condition that appears to be fatal. CAD and ischemic cardiac damage are the main causes of SCD. In these casesin which the death occurs within six hours from the onset of ischemic damage, the histological myocardial changes are not specific and cannot provide clear evidence for the postmortem diagnosis.

In recent years, various immunohistochemical studies for the detection of early myocardial infarction were conducted, and various markers were analyzed.[1,2] In fact, following the ischemic injury to the muscle tissue of the heart, the biomarkers released by the damaged cells and the altered cellular and extracellular molecules normally expressed in cardiac tissue can be detected microscopically using immunohistochemical techniques.

This study highlights the utility of dystrophin as a marker of early ischemic damage in a sample of SCDs with macroscopic and microscopic evidences of cardiovascular disease. Dystrophin was isolated in cardiac muscle, tightly associated oligomeric complex of proteins known as dystrophin-glycoprotein complex that plays an important mechanical function in stabilizing the sarcolemma during cardiac contraction and in the transmission of myofibers contraction force. Dystrophin may prove to be a very useful marker in detection of ischemic lesions utilizing immunohistochemical staining of the cardiac tissue, especially in cases in which the conventional histology, with hematoxylin-eosin, fails.

The study reveals that as a result of cellular ischemic damage the progressive loss of sarcolemmal staining of dystrophin occurs. The damage on the tissue is evidenced in microscopic areas with partial loss, such as interrupted sarcolemmal staining of dystrophin in cardiac myocytes, and in microscopic areas with complete loss of staining. These results show a time-dependent depletion of dystrophin; in fact, there is an increase in the area of marker depletion with the increase of post-ischemic time.

In conclusion, the present study demonstrates the usefulness of the dystrophin as marker of early ischemic damage. This marker can be a useful tool to reveal, with the immunohistochemical method, the myocardial damage within six hours of the onset of ischemic injury.

Reference(s):

1. Ortmann C., Pfeiffer H., Brinkmann B. A comparative study on the immunohistochemical detection of early myocardial damage. *Int J Legal Med*. 2000; 113(4):215-20.
2. Campobasso C.P., Dell'Erba A.S., Addante A., Zotti F., Marzullo A., Colonna M.F. Sudden cardiac death and myocardial ischemia indicators: a comparative study of four immunohistochemical markers. *Am J Forensic Med Pathol*. 2008; 29(2):154-61.

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