A case report of giant cell myocarditis diagnosed on autopsy.

Maria S. Madentzoglou
Department of Clinical Pathology, Linköping University, 581 85 Linköping, Sweden
email: nebulaaluben@yahoo.gr

Background & objective

Giant cell myocarditis is an unusual cause of death which often presents as rapidly evolving congestive heart failure. Individuals of young age are usually affected. Etiologically, it is possibly autoimmune related. Its prognosis is poor. Macroscopically heart is often hypertrophic, dilated with focally pallor and scarring. Histologic findings include myocardial necrosis, multinucleated giant cells, and mixed inflammatory cell infiltrate [1–2].

A case report of giant cell myocarditis diagnosed on autopsy presents briefly.

Methods

A 68-year-old man who lived alone, with tablet-treated hypertension, was found dead at home, at the bathroom, after not having contact with his relatives for a few days. Circumstances of death raised no suspicion of crime, abuse, or suicide. Clinical autopsy was conducted to determine the cause of death.

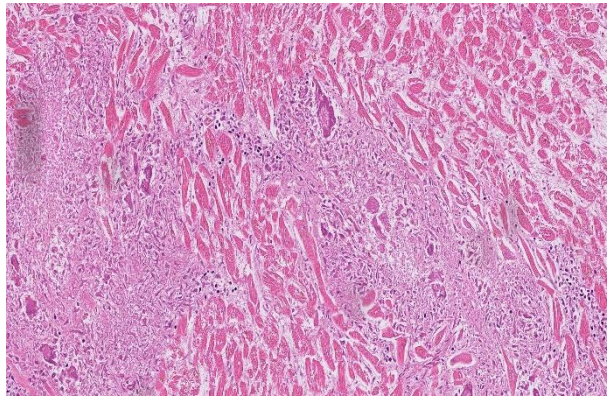

Figure 1. Haematoxylin-Eosin, X200, heart with multinuclated giant cells.

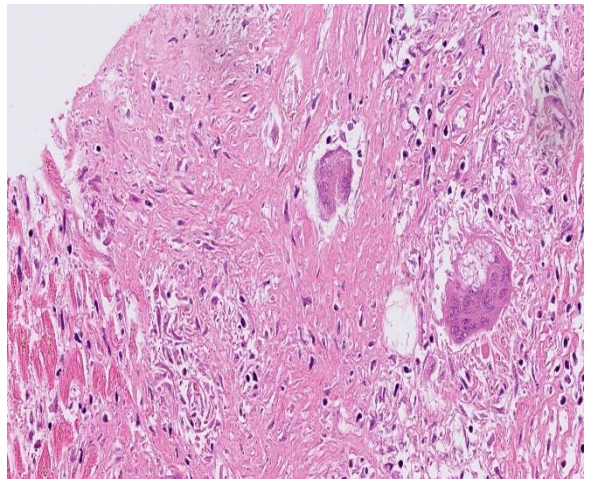

Figure 2. Haematoxylin-Eosin, X400, heart with multinuclated giant cells.

Results

External body inspection revealed no injuries or scars. The deceased hade normal body constitution. Cyanosis on the nails was noted.

The findings of internal organs examination were the following: Heart with symmetrical hypertrophy, weight 540gr. Small isolated whitish areas frontal and lateral in the right ventricle. Mild but not critical coronary atherosclerosis only focally. Regarding the other organs, mild pulmonary oedema, a few simple kidneys cysts, enlarged prostate without tumour and generalized moderate atherosclerosis were noticed.

Microscopic examination revealed myocardium hypertrophy, perivascular fibrosis and focal myofiber break up. (Fig.1-2)

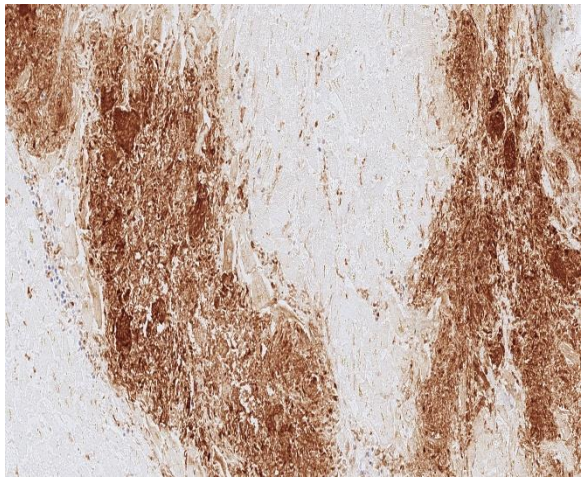

Figure 3. Immunohistochemical staining with CD68, X200, heart with multinucleated giant cells.

Foci with fibrosis, sparse mixed inflammatory cells, and necrotic myocardial fibres with the presence of multinucleated giant cells were found. No vasculitis, no foreign material, and no evidence for systematic granulomatous or other inflammation or sarcoidosis were described. (Fig. 1-2)

Immunohistochemical staining with CD68 in representative tissue sampled from the heart was strongly positive in multinucleated giant cells.(Fig.3) Furthermore, immunohistochemical staining with CD4, CD8 were focal weak positive only, but tissue material was partially autolytic.

Immunohistochemical staining with Desmin was also conducted with very weak positivity only focally, which was difficult to evaluate and partially not reliable due to autolytic material. (Fig. 4)

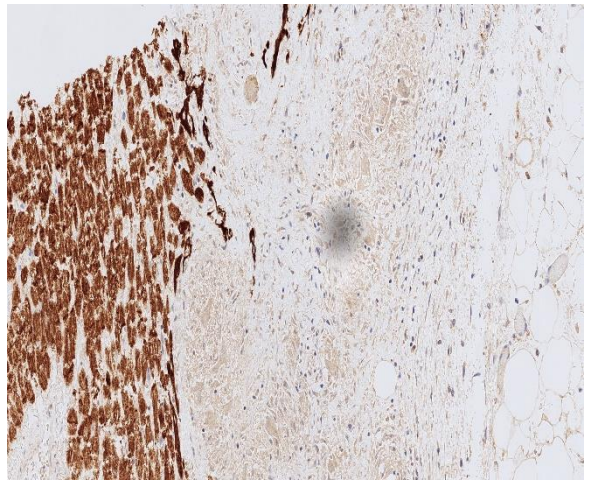

Figure 4. Immunohistochemical staining with Desmin, X200, heart with multinucleated giant cells.

Conclusion

Autopsy's findings together with histological findings of heart described above, with multinucleated giant cells without necrosis, gave rise to suspicion of giant cell myocarditis, an unusual cause of death. The aetiology is unclear, autoimmune idiopathic, drug-associated, or viral cause have been described [1–2]. In addition, multinucleated giant cell formation can also occur rarely in myocardial infarction. Desmin immunohistochemical staining is then positive in the multinucleated giant cells supporting multinuclear myogenic giant cell formation under myocardial infraction[1–2].

In this case myocardial infraction considered less likely due to the absence of critical stenos in coronary arteries. Giant cell myocarditis was determined to be the most likely cause of death in this case.

References:

1. **Xu J, Brooks EG. Giant Cell Myocarditis: A Brief Review. Arch Pathol Lab Med. 2016 Dec;140(12):1429-1434. doi: 10.5858/arpa.2016-0068-RS. PMID: 27922771.**
2. **Leone O, Pieroni M, Rapezzi C, Olivotto I. The spectrum of myocarditis: from pathology to the clinics. Virchows Arch. 2019 Sep;475(3):279-301. doi: 10.1007/s00428-019-02615-8. Epub 2019 Jul 11. PMID: 31297595.**