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The Mummified Heart: A Problem in Medicolegal Diagnosis

An occasional problem encountered in forensic medicine is the finding of a mummified body, preserved by chance in an environment conducive to desiccation. Such individuals have usually died suddenly in an obscure location, precluding their discovery and allowing time for natural mummification to take place. In the absence of evidence of trauma, it is important to establish a natural disease process such as arteriosclerotic heart disease (ASHD) to account for death.

It is possible to dissect such mummified cadavers, rehydrate the tissues, and examine them microscopically [1]. The distinction of the artifacts induced by desiccation and rehydration from pathologic change is important. To this end, an experimental study was undertaken of the changes seen in mummified normal [2] and pathologic [3] tissues. Examination of the cardiovascular system bears directly on the problem.

Methods and Materials

The tissues studied were obtained from 37 adult human cadavers undergoing autopsy, all within 24 h after death. Specimens measuring approximately 1 cm³ were taken from areas showing evidence of acute myocardial infarction or scarring and from areas of coronary atherosclerosis and thrombosis. Sections of congested lung, liver, and spleen were also selected.

Each specimen was bisected. One half was fixed immediately in 10% formalin and processed normally. The other half was placed in an Elconap oven at 40°C (104° F) for 7 to 14 days. After this period, the specimens were dry, dark brown, and reduced to approximately 10% of their original weight. The specimens were rehydrated with Ruffer's solution, 50 parts of water, 30 parts of absolute alcohol, and 20 parts of a 5% sodium carbonate solution [4]. After immersion for 24 to 48 h, the rehydrated specimens were fixed in absolute alcohol for 24 h, embedded in paraffin, and processed normally. Staining of the sections was according to standard techniques [5], and included hematoxylin and eosin (H&E), Masson's trichrome, Mallory's phosphotungstic acid-hematoxylin (PTAH), and the Gomori iron stain.

To limit observer bias the slides (part of a more extensive study considering many other disease processes) were coded numerically and not examined until several weeks or months had elapsed. The slides of the mummified tissue were examined first and then compared to the fresh tissue slides.

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Results

Examination of a severely atherosclerotic and thrombosed coronary artery showed excellent preservation of the atherosclerosis and calcification after mummification and rehydration. The thrombus was reduced to eosinophilic material indistinguishable with H&E from a mummified blood clot. The PTAH stained the material within the lumen a diffuse blue. The red blood cells and fine detail of the fibrin were no longer seen, but the mesh-like fibrin pattern remained (Fig. 1).

Myocardial interstitial fibrosis was recognizable, especially with the trichrome stain. In contrast, an acute myocardial infarct was undiagnosable, the mummified necrotic muscle being indistinguishable from the adjacent autolyzed myocardium. Only a few fragmented neutrophils of the inflammatory infiltrate were tentatively identified.

A healing myocardial infarct showed a large area of fibrosis with peripheral organization by granulation tissue. With mummification, the fibrosis was seen as a lighter-staining eosinophilic area with a decreased number of nuclei. Again, trichrome staining showed the fibrosis very well (Fig. 2), and the peripheral organization was also clear.

Chronic passive congestion of the lungs, liver, and spleen was well preserved, particularly the hemosiderin pigment present, which retained a marked avidity for the iron stain.

Discussion

A previous study [2] has demonstrated good preservation of mummified normal myocardium. The current study shows variable preservation of pathologic changes in the myocardium. The necrosis of muscle in an acute infarct being in essence a process of in-situ autolysis, an infarcted area cannot be distinguished from adjacent autolyzed myocardium in a mummified heart. Diagnosis of an acute infarct in a mummified heart would have to be based on the finding of the remains of a neutrophilic infiltrate, a problematic matter at best in the experimental setting and probably impossible in a naturally preserved mummy.

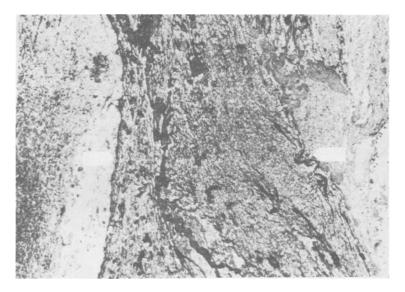


FIG. 1—Mummified coronary thrombosis. The fibrin meshwork of the thrombus (between the arrows) retains its affinity with the PTAH stain, but the erythrocytes have autolyzed (magnification, approximately $\times 80$).



FIG. 2—Mummified healed myocardial infarct. There is excellent preservation of reparative fibrous tissue (the central lighter staining area) and adjacent regenerative myocardium (trichrome; magnification, approximately $\times 80$).

The fibrosis of a healed infarct should remain detectable, as should the evidence of chronic passive congestion resulting from a failing cardiovascular system. Specific stains for fibrous tissue and iron are of value in demonstrating these conditions.

Generalized atherosclerosis [6, 7] and coronary artery disease [8, 9] are readily demonstrable in actual mummies. The results of this experimental study are in accordance with the paleopathologic evidence. Coronary thrombosis should also remain a detectable lesion in the mummified heart when the appropriate PTAH stain is used.

In conclusion, the diagnosis of an acute myocardial infarct as a cause of death is probably not possible in a mummified body. Strong presumptive evidence, including coronary artery disease or thrombosis, myocardial scarring, and visceral congestion, should be preserved and allow for the assignment of ASHD as a cause of death in cadavers found mummified.

Summary

Bodies that have been discovered in a naturally mummified state are occasionally presented to forensic practitioners for the determination of cause of death. An experimental study of mummification has been presented that suggests that while the diagnosis of acute myocardial infarct is probably not possible under these conditions, strong presumptive evidence such as coronary artery disease or thrombosis, myocardial scarring, and chronic visceral congestion is well preserved. Such findings would allow the assignment of arteriosclerotic heart disease as a cause of death in mummified cadavers.

References

- [1] Sandison, A. T., "The Study of Mummified and Dried Human Tissues," in Science in Archaeology, 2nd ed., D. Brothwell and E. Higgs, Eds., Praeger, New York, 1970, pp. 490-502.
- [2] Zimmerman, M. R., "Histological Examination of Experimentally Mummified Tissues," American Journal of Physical Anthropology, Vol. 37, No. 2, Sept. 1972, pp. 271-280.

- [3] Zimmerman, M. R., "An Experimental Study of Mummification Pertinent to the Antiquity of Cancer," Cancer, Vol. 40, No. 3, Sept. 1977, pp. 1358-1362.
- [4] Ruffer, M. A., Studies in the Paleopathology of Egypt, University of Chicago Press, Chicago, 1921.
- [5] Luna, L., Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology, McGraw-Hill, New York, 1968.
- [6] Cockburn, T. A., Barroco, R., Reyman, T. A., and Peck, W., "Autopsy of an Egyptian Mummy,"
- Science, Vol. 187, No. 4182, 28 March 1975, pp. 1155-1160.
 [7] Sandison, A. T., "Degenerative Vascular Disease," in Diseases in Antiquity, D. Brothwell and A. T. Sandison, Eds., Charles C Thomas, Springfield, Ill., 1967, pp. 474-488.
- [8] Zimmerman, M. R. and Smith, G. S., "A Probable Case of Accidental Inhumation of 1600 Years Ago," Bulletin of the New York Academy of Medicine, Vol. 51, No. 7, July-Aug. 1975, pp. 828-837.
- [9] Long, A. R., "Cardiovascular Renal Disease: Report of a Case Three Thousand Years Ago," Archives of Pathology, Vol. 12, No. 1, July 1931, pp. 92-94.

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