ELSEVIER

Cardiovascular Pathology 21 (2012) e31 – e32

CARDIOVASCULAR PATHOLOGY

Correspondence

Sudden death and coronary intimal thickening

To the Editor:

I read with great interest the report of Tavora et al. [1], who accurately studied sudden coronary death caused by pathologic intimal thickening without atheromatous plaque formation. In a large institution, over 300 hearts from patients experiencing a sudden unexpected death were studied retrospectively; the authors found that nonatheromatous atherosclerosis accounts for over 10% of sudden coronary death and is significantly more frequent at younger age, in female gender, and in black race [1].

Although I fully agree with the authors' results and conclusions, I would like to place them into the larger context of ongoing investigation on the coronary artery plaques that have been reported to be already present in fetuses and infants [2–5].

Among 22 fetuses dying suddenly and unexpectedly after 35 weeks of gestation, in 12 (55%) cases, multifocal structural alterations of all the coronary arteries were reported, and these were more severe along the anterior descending branch of the left coronary artery [2]. In seven (32%) of these fetuses, foci of altered architecture of the media with thinning and fiber fragmentation were observed, even in fields far from the bifurcations. The smooth muscle cells (SMCs) showed loss of polarity, forming columns of myocytes located perpendicular to the axis of the media itself and infiltrating the subendothelial connective tissue. In five (23%) additional fetuses, besides this intense reaction of the SMCs of the media, increased amounts of mucoid ground substance were observed in the subendothelial connective tissue, with formation of intimal lesions of proliferative aspect (Fig. 1A, B). Such processes also seem to determine fragmentation and detachment of the internal elastic membrane [2].

Histological study of the coronary arteries of the 36 infants having died suddenly and unexpectedly showed thickening of varying severity in the artery walls in 22 cases (61%). A significant correlation was evident between intimal thickening and parental cigarette smoking ($P<.05$). Marked cell proliferation was present. The proliferating cells were arranged in columns with the axis perpendicular to the tunica itself. These elements infiltrated the intima together with acid mucopolysaccharide deposits, consisting of type A and C chondroitin sulfates and hyaluronic acid, probably synthesized by the SMCs themselves. Low numbers of monocytes were present; B lymphocytes were generally absent [4,5].

The histological study of the cardiac conduction arteries of 70 infants dying suddenly and unexpectedly showed thickening of the sinoatrial node and/or atrioventricular artery associated with a thickening of varying severity in coronary artery walls in 15 cases (21.4%). The cardiac conduction system was removed in two blocks for paraffin embedding and serially cut. The lesions were marked by thickening and deposits of amorphous material and mainly lipids in the intima, as well as fragmentation of the elastic fiber system. A significant correlation was evident between early atherosclerotic lesions and parental cigarette smoking ($P<.05$) [3].

The author is convinced that the postmortem examination of the cardiac conduction system and its arteries on serial sections plays a fundamental role in the identification of the conclusive causes of sudden cardiac death in the absence of atheromatous plaque formation, especially in young subjects. Nevertheless, it would be essential that a new formulation of the study approach in young subjects should include the postmortem examination of the cardiac conduction system and its arteries.

In conclusion, besides the anatomopathological findings of atheromatous and nonatheromatous atherosclerosis, the initial stages of atherosclerotic lesions in the coronary arteries and in the arteries of the cardiac conduction system in early stages of human development needs further study and consideration.

Giulia Ottaviani
Dipartimento di Scienze Materno-Infantili
Università degli Studi di Milano
Milan, Italy
E-mail addresses: giulia.ottaviani@unimi.it,
http://www.users.unimi.it/giuliaottaviani/

doi:10.1016/j.carpath.2011.07.003

Competing interest statement: No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article. In particular, there are no competing interests related to this article. The author has no conflict to disclose.

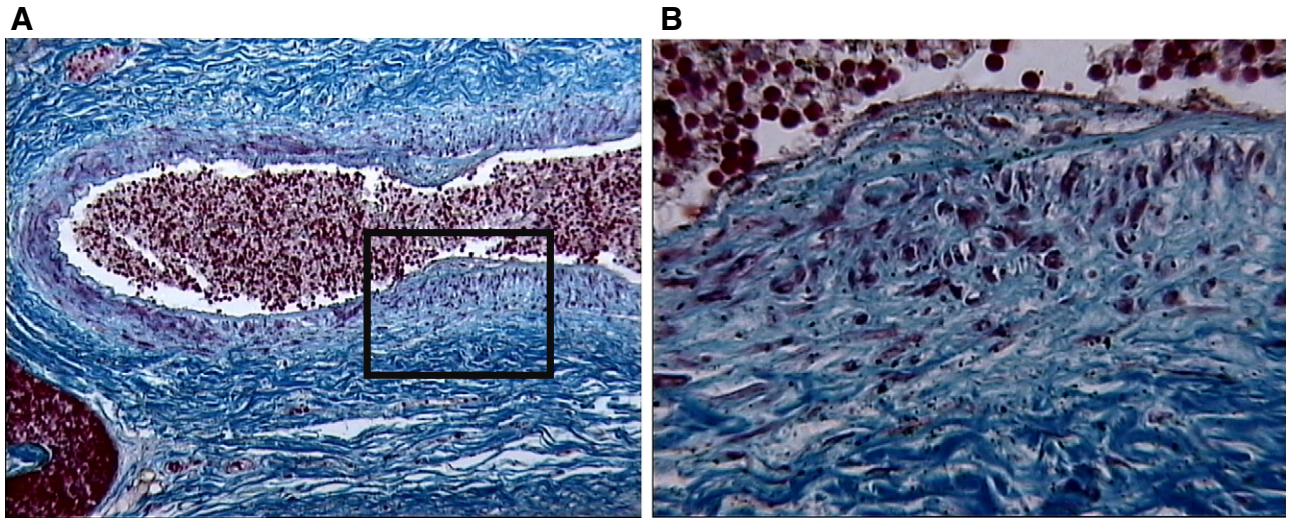

Fig. 1. Right main coronary artery of a male fetus who died suddenly and unexpectedly at the 37 weeks' gestation. The myointimal thickness shows increased amounts of mucoid ground substance. Azan stain; original magnification: (A) ×100, (B) ×400.

References

[1] Tavora F, Cresswell N, Li L, Ripple M, Fowler D, Burke A. Sudden coronary death caused by pathologic intimal thickening without atheromatous plaque formation. Cardiovasc Pathol 2011;20: 51–7.

[2] Matturri L, Lavezzi AM, Ottaviani G, Rossi L. Intimal preatherosclerotic thickening of the coronary arteries in human fetuses of smoker mothers. J Thromb Haemost 2003;1:2234–8.

[3] Matturri L, Ottaviani G, Lavezzi AM, Rossi L. Early atherosclerotic lesions of the cardiac conduction system arteries in infants. Cardiovasc Pathol 2004;13:276–81.

[4] Ottaviani G. Crib death. Sudden unexplained death of infants: The pathologist's viewpoint. Berlin Heidelberg, Germany: Springer-Verlag; 2007.

[5] Milei J, Ottaviani G, Lavezzi AM, Grana DR, Stella I, Matturri L. Perinatal and infant early atherosclerotic coronary lesions. Can J Cardiol 2008;24:137–41.