General and site-specific features of atherosclerosis

Søren Dalager, MD



This PhD dissertation was accepted by the Faculty of Health Sciences, University of Aarhus, and defended on May 31, 2007.

Official opponents: Ulrik Baandrup, Marie-Louise M. Grønholdt, and Karen Ege Olsen.

Supervisors: William P. Paaske, Erling Falk, and Ingrid Bayer Kristensen.

Correspondence: Søren Dalager, Sletterhagevej 49, 2. th., 8240 Risskov, Denmark.

E-mail: soren@dalager.eu

Dan Med Bull 2007;54:236

ABSTRACT

The work was carried out at the Department of Cardiothoracic and Vascular Surgery T and the Department of Cardiology B, Aarhus University Hospital, Skejby, and the Institute of Forensic Medicine, University of Aarhus.

The PhD dissertation deals with histopathologic examination of atherosclerosis in the coronary arteries, the carotid arteries, and the superficial femoral arteries. The arteries were from a large forensic autopsy material (100 individuals, 4756 paraffin blocks).

The aims were examination of atherosclerosis expression and development in different arteries. The findings were related to research and prevention. More specifically, we focused on identification of asymptomatic persons at high risk of complications, in our case sudden death caused by coronary atherosclerosis.

First, we found that atherosclerosis was more common and developed earlier in some arteries (the coronary arteries and the carotid bifurcation) than others (the femoral arteries). The composition also differed, some arteries had many foam cell lesions and lipid core plaques (the common carotid arteries and the carotid bifurcation) while others had more fibrous plaques and few foam cell lesions (the femoral arteries).

Next, we found that presence of plaque in the femoral arteries was more informative than carotid plaque presence and at least as informative as coronary plaque presence in the identification of those individuals who had coronary deaths.

Finally, we examined whether measures of plaque inflammation provided additional diagnostic information over that of plaque size alone in the identification of individuals who died from coronary causes. We found that inflammation cannot generally be anticipated to contribute to identification of individuals at risk. Only adventitial lymphocyte infiltration seemed to have some potential.

In conclusion, our results point to an artery-specific atherogenesis and highlight the importance of intensive medical treatment of patients with lower-extremity arterial disease because these patients have even more advanced disease in the coronary and carotid arteries.