PANARTERITIS PRECIPITATING EXTENSIVE CIRCUMFERENTIAL ACUTE MYOCARDIAL INFARCTION. A CASE REPORT

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It is widely known that other causes than recent coronary thrombosis may precipitate acute myocardial infarction in the presence of coronary atherosclerosis.

A 48 year old male patient was admitted due to acute coronary insufficiency. the ECG showed anterolateral necrosis and lateral ischemia. Despite medication angina persisted and he died immediately after coronary angiography. At autopsy, established coagulation necrosis was observed in the internal half and the subendocardium of the lateral and posterior walls, of the left ventricle. Early coagulation necrosis occupied the inner half of the anterior, posterior and septal walls. Severe atherosclerotic coronary lesions were found in all major coronary trunks. An extensive panarteritis, involving extra and intramyocardial branches, consisting of mononuclear cells and prominent edema, was observed. A mixed mechanism may be invoked to explain the extensive myocardial necrosis: panarteritic infiltrates and extensive edema and humoral-induced coronary spasm.

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The issue of wheter coronary thrombosis precedes and initiates acute myocardial infarction (AMI) has come to the boiling point in the recent past[1]. Reports from our laboratory[2] have confirmed previous observations[1-3] on the abscence of recent coronary thrombosis in some cases of AMI, and it is widely known that several non-atherosclerotic factors may precipitate AMI in the presence of severe coronary atherosclerosis.

We report on a patient in whom panarteritis precipitated extensive circumferential AMI.

CASE REPORT

A 48 year old male was admitted for several effort-related episodes of substernal pain over the last month. After an episode at rest, he came to the emergency room. His blood pressure was normal and an S4 could be heard at the apex. The initial ECG showed left ventricular hypertrophy, an anterolateral AMI and lateral ischemia. He was treated with oral nitrates and nifedipine, 40 mg/day. Despite intravenous nitro glycerin, chest pain persisted. An emergency coronary angiogram was perfomed, after which he complained of severe chest pain. The ECG showed electromechanical dissociation and the patient died.

At autopsy, the internal half and the subendocardium of the lateral and posterior walls showed gross coagulation necrosis (fig. 1). Early necrotic areas occupied the inner half of the anterior, posterior and septal walls, where extensive subendocardial myocytolysis was also observed. The damage myocardium comprised 61% of the ventricular areas. The stenosis of the four major coronary vessels, considered as a whole, was 90 ± 5.4% of their lumen (fig. 2). No recent coronary thrombi were found. The most striking finding was an inflamatory infiltrate involving all three layers of all the coronary tree (fig. 3). The arteritis involved extra and intramyocardial branches, and consisted of mononuclear cells, scattered eosinophils and neutrophils. Prominent edema as well as the infiltrate extended through the full thickness of the vessel walls and the perivascular space (fig. 4). The areas of edema did not stain for fibrin. No necrotizing vasculitis was observed. The endothelium appeared undamaged. All lesions were in similar stages of development and healing lesions were scarce.

DISCUSSION

Although these microscopical findings are similar to drug-induced vasculitis[4], a close relationship bet-

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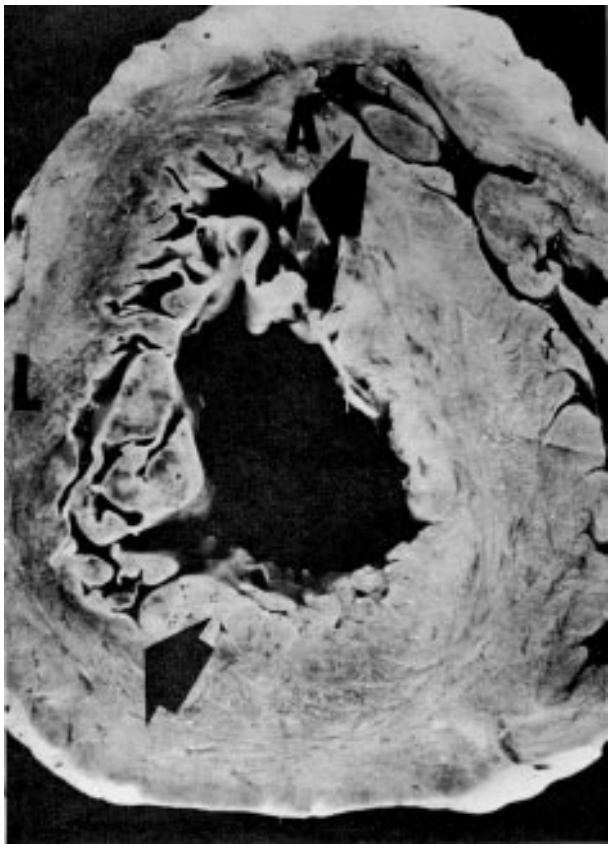

Fig. 1 - Gross aspect of the left ventricle. Extensive necrosis is shown between arrows. A: anterior wall, L: lateral wall.

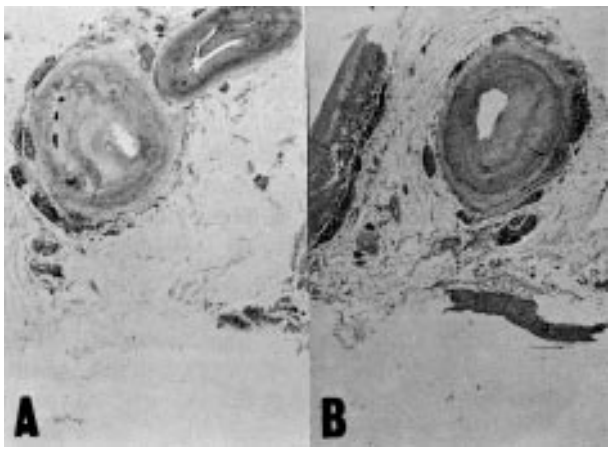

Fig. 2 - A: Right coronary artery. B: Left coronary artery. Panarteritis of small branches surrounding the major trunks. H-E X 7.

ween them and drugs or autoinmune diseases could not be demonstrated. Anyway, the patient suffered from an acute respiratory illness in his rural habitat, and he could presumably had taken penicillin, the largest single class of drug associated to vasculitis[4].

Our patient had an AMI precipitated by an extensive panarteritis superimposed on a severe coronary atherosclerosis. AMI due to coronary arteritis has, as in this case, inflammatory infiltrates and edema and fibrinoid necrosis and/or giant cells, according to

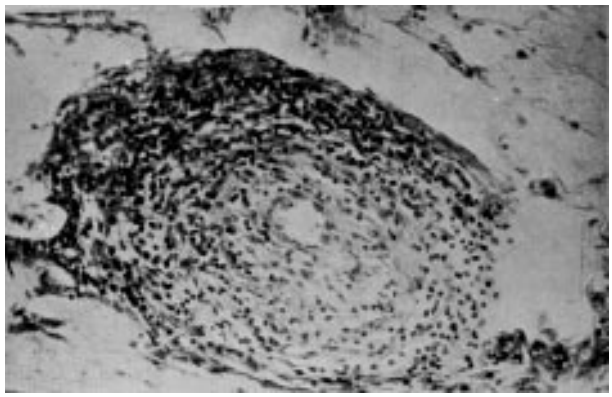

Fig. 3 - An inflammatory infiltrate involving all three layers of a minor coronary artery. H-E X 100.

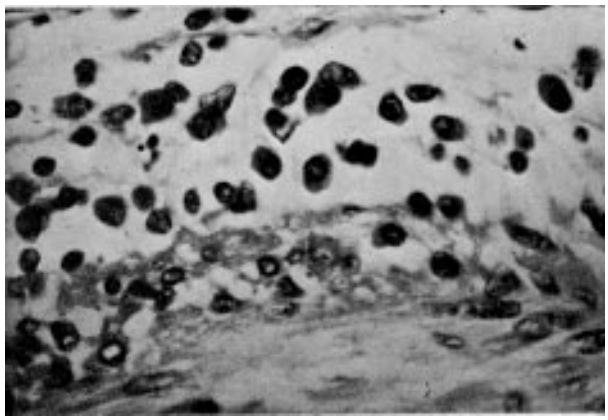

Fig. 4 - Prominent edema and inflammatory infiltrate of plasmocytes, mast cells, hystiocytes and lymphocytes in the arterial wall. H-E X 100.

each disease. Then, increasing edema and infiltrates on an atherosclerotic artery may totally occlude the vessel. However, it cannot be ruled out that local release of substances such as serotonin[6], secreted by mast cells of by the damaged wall (prostaglandins) may precipitate spasm and subsequent AMI.

In our patient, a mixed mechanism may be suspected: panarteritic infiltrates and extensive edema, and humoral-induced coronary spasm. Accordingly, as lesions do not have a vascular topography and damaged myocardium showed both, early and established necrosis, the circumferential involvement may have resulted from different mechanisms at different stages.

RESUMO

Sabe-se que, além aa trombose coronária, outras causas podem precipitar infarto agudo do miocárdio em presencça de aterosclerose coronária. Um paciente com 48 anos de idade foi internado devido a insuficiência coronária aguda. O eletrocardiograma mostrou necrose ântero-lateral e isquemia lateral. A des-

peito da medicação a angina persistiu e o paciente faleceu imediatamente após a angiografia coronária. À necrópsta, observou-se necrose de coagulação estabelecida na metade interna e no subendocárdio das paredes lateral e posterior do ventrículo esquerdo, e necrose de coagulação recente nas paredes anterior, posterior e septal. Em todas as artérias coronárias principais havia lesões ateroscleróticas severas. Observou-se também panarterite difusa, envolvendo ramos coronários extra e intramiocárdicos, consistindo de células mononucleares e edema acentuado. Um mecanismo misto pode ser invocado para explicar a extensa necrose miocárdica: infiltrados panarteriais, edema extenso e espasmo coronário induzido por fatores humorais.

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