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Dissecting Aneurysm of Coronary Artery Associated with Drug Abuse and Pulmonary Lesions

The rare isolated dissecting aneurysm of a coronary artery (DACA), known to be causally related to trauma and Marfan's syndrome, has also been linked with pregnancy and the postpartum state [*1*]. Hormonal effects on the ground substance or connective tissue of the coronary vessels have been invoked to explain this phenomenon [*2*]. The well-known relationship of dissecting aneurysms of the aorta with cystic medial necrosis prompted a search for the association of this degenerative change with dissecting aneurysms of the coronary artery. Various authors have confirmed this association [*3,4*].

This report describes a dissecting aneurysm of the right coronary artery associated with cystic medial necrosis of the vessel discovered at the necropsy of a 32-year-old black man with evidence of intravenous drug abuse. Although the gross and microscopic pulmonary findings were compatible with heroin-induced pulmonary edema as the cause of death, the significance of the right coronary artery dissection must be carefully assessed. Moreover, the possible association between intravenous drug abuse and dissection of the coronary artery invites speculation about the possible etiology of this unusual vascular lesion.

Report of a Case

The patient was a 32-year-old black man who was found pulseless at home in the bathroom. Local police brought him to the University of Michigan Medical Center for necropsy after he was pronounced dead on arrival at a neighboring hospital.

Only scanty information could be obtained from family members concerning the circumstances of his death. A syringe and needle were found in a household cupboard by police investigating his death.

At necropsy, linear scars interpreted as "tracks" were noted in both his antecubital fossae. A knotted strip of cloth thought to be a tourniquet was found inside a pants leg. He had none of the external manifestations of Marfan's syndrome such as arachnodactyly or unusual height. Gross examination of the heart revealed that the right coronary artery was nearly totally occluded by a dissecting aneurysm located 2 cm from the origin of the vessel. The occluded segment was only 4 mm in length. There was no hemorrhage in the epicardial adipose tissue overlying the dissection. The left coronary artery was grossly normal in its entirety. No dilation of the cardiac chambers was present nor was

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there gross evidence of hemorrhage, necrosis, or scarring of the myocardium. The cardiac valves were grossly unremarkable.

Microscopic examination of the right coronary artery at the site of the dissection revealed a cleavage plane involving both the media of the vessel as well as the elastica interna (Fig. 1). The bulging, greatly thickened intima with the adjacent media almost

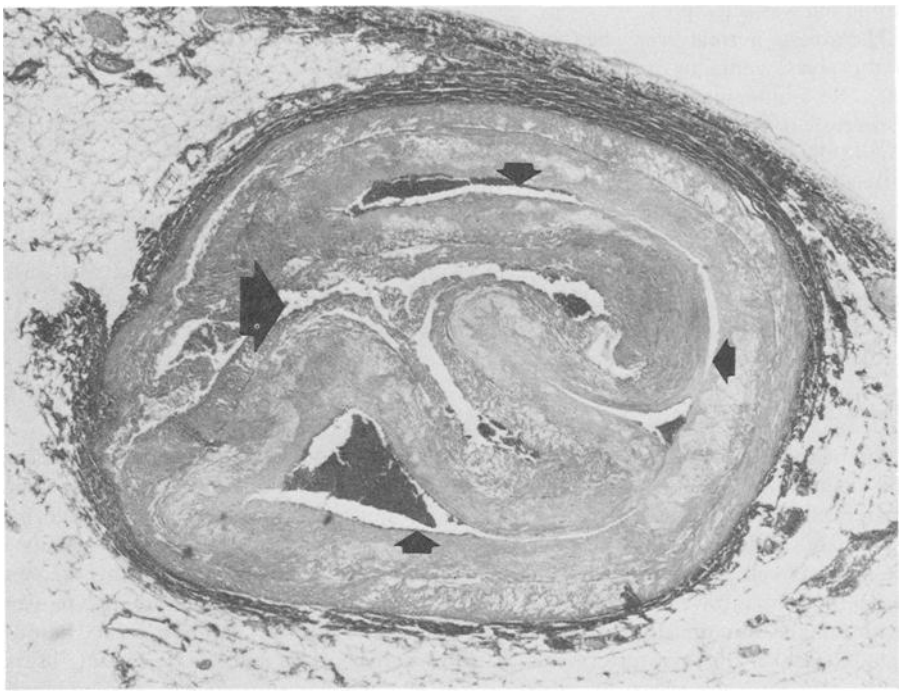

FIG. 1—*Section of right coronary artery with dissecting aneurysm (true lumen at small arrows, dissection at large arrow) cystic medial necrosis, and faintly visible fragmented internal elastic lamina (elastic—van Gieson, ×15).*

totally occluded the true lumen of the vessel. The overall histologic appearance of the vessel was extremely bland due to the total absence of intramural hemorrhage or inflammation.

The thickened intima and media of the right coronary artery in the region of the dissection contained confluent vacuoles filled with mucoid material identified as acid mucopolysaccharide by special stains. The elastica interna was fragmented and discontinuous. These degenerative changes were interpreted as cystic medial necrosis.

Step sections of the affected coronary artery were studied for evidence of occlusion of the vasa vasorum by granulomas or thromboemboli without success.

The proximal aorta was grossly normal and without evidence of dilatation or dissecting aneurysm.

Grossly, the lungs were massively edematous, congested, and hemorrhagic, weighing 1700 g combined. The pulmonary arteries showed no atherosclerosis.

Microscopically, the lungs manifested severe pulmonary edema, congestion, and intra-alveolar hemorrhage. Occasional scattered pigment-laden macrophages were present in

alveoli, but there was no evidence of acute or organizing pneumonia. The pulmonary arteries were thickened and tortuous. There were scattered "angiomatoid" formations involving pulmonary arterioles (Fig. 2). Rare discrete sclerotic granulomas were also noted in the lung parenchyma (Fig. 2). Within these granulomas as well as in alveolar septa were small aggregates of birefringent crystalline material interpreted as talc (magnesium silicate) crystals. No specific identification of the crystalline material by X-ray diffraction was attempted.

The grossly normal liver when examined microscopically revealed a diffuse infiltration of the portal zones by lymphocytes, plasma cells, and rare polymorphonuclear leukocytes. No granulomas accompanied this liver "triaditis" nor was there histologic evidence of necrosis or collapse of hepatic parenchyma. There was no enlargement of lymph nodes around the liver hilum or in the rest of the abdomen.

Blood, urine, gastric contents, bile, liver, kidney, and brain were submitted to the Michigan Department of Public Health Crime Laboratory for toxicologic analysis. Qualitative analysis of the urine by thin-layer chromatography revealed the presence of morphine and procaine but failed to detect amphetamines. Analysis of blood showed the presence of 0.11% ethyl alcohol but did not detect barbiturates or procaine. The submitted bile and organs were not analyzed. Analysis of the contents of the syringe and needle failed to detect the presence of controlled substances.

Discussion

The association of intravenous drug abuse, sudden death, severe pulmonary edema and hemorrhage, and DACA with cystic medial necrosis raises two intriguing issues. First of all, had the subtle coronary artery lesion not been discovered, death would have been readily ascribed to heroin-induced pulmonary edema. The presence of morphine in the urine as well as the gross and microscopic pulmonary findings support this diagnosis of death from intravenous narcotism [*5*]. The relationship of DACA to the patient's death remains obscure. DACA more commonly involves the left coronary artery than the right. Review of the literature reveals that obstruction of the anterior descending branch of the left coronary artery by an aneurysm commonly results in sudden and abrupt death. DACA of the right coronary artery more commonly results in protracted symptoms and myocardial infarcts, although sudden death with obstruction of this vessel is not unknown [*6, 7*]. The pulmonary changes induced by a failing ischemic heart could easily mimic heroin-induced edema and hemorrhage.

Secondly, the combination of DACA with pulmonary vascular lesions invites speculation concerning the pathogenesis of the coronary artery dissection. Angiomatoid conversion of pulmonary arterioles and talc granulomas are common and well described in the lungs of drug addicts [*8, 9*]. Talc is commonly used as filler in medications manufactured for oral use and also, incidentally, to adulterate heroin, and may thus be injected when tablets are dissolved and taken parenterally. The talc crystals are known to induce thrombosis and occlusion of arterioles and capillaries as well as perivascular and interstitial granulomas. Angiomatoid formation, seen to a striking degree in the patient described, is the net result of vascular occlusion and recanalization.

Brody et al [*6*], in their study of 18 patients with DACA, noted that 3 of the 12 cases unrelated to pregnancy had severe pulmonary disease and postulated that hypoxia may have "augmented the production of dissecting aneurysms." Although there is no clinical documentation, the pulmonary lesions described in this patient would probably result in respiratory pathophysiologic change, a not uncommon finding in drug addicts [*10*].

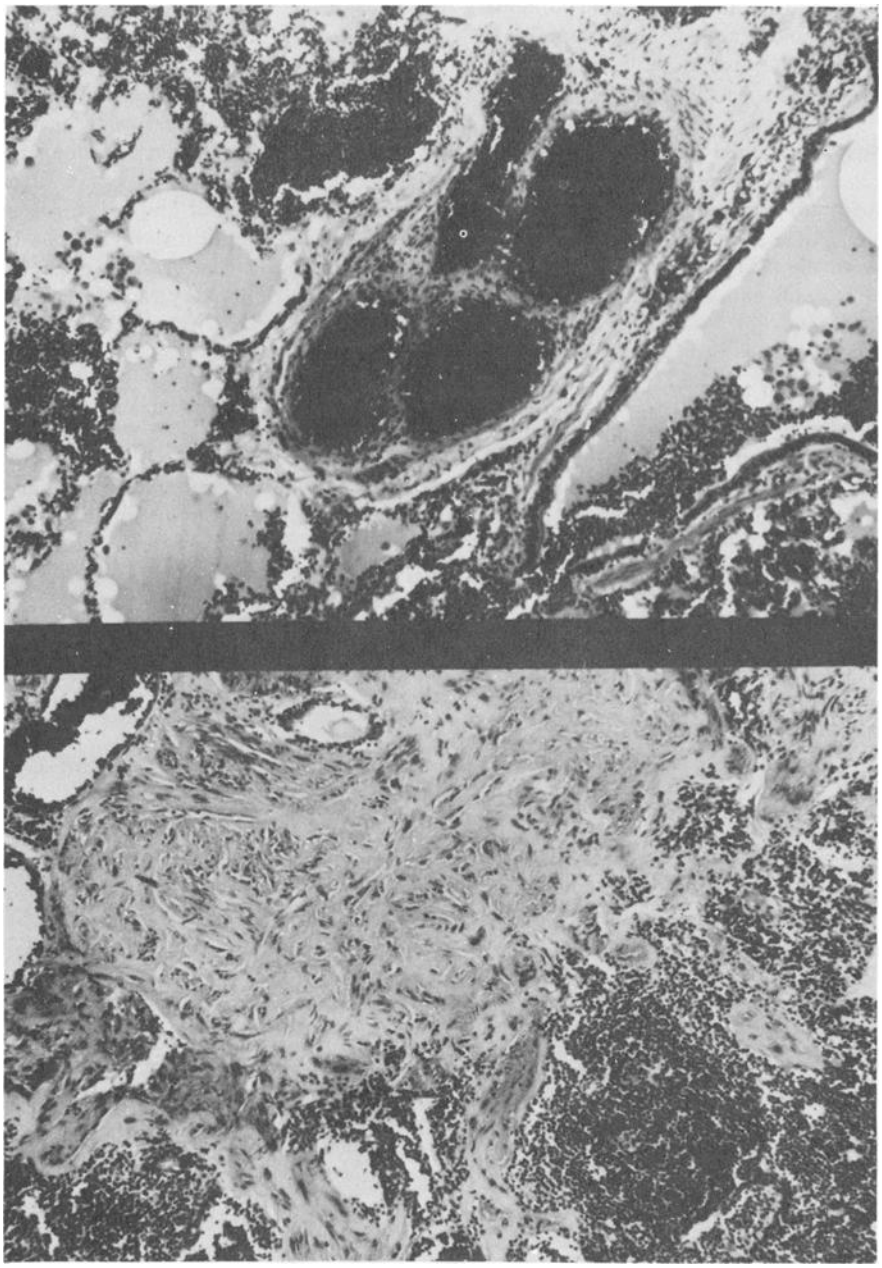

FIG. 2—(left) *Sclerotic pulmonary granuloma with adjacent hemorrhage (hematoxylin and eosin, ×160) and* (right) *angiomatoid conversion of pulmonary arteriole with pulmonary edema and intra-alveolar hemorrhage (hematoxylin and eosin, ×160).*

Tredal et al [*11*] have recently noted an association between cystic medial necrosis of the pulmonary artery and aorta and pulmonary hypertension. Pulmonary hypertension has been described in drug addicts secondary to vascular embolic occlusion with recanalization [*12,13*]. The diagnosis of pulmonary hypertension per se could not be supported in this patient on morphologic grounds.

The pulmonary vascular lesions seen in this patient are undoubtedly the direct consequence of intravenous drug abuse. Moreover, it is suggested that there may be an association between these pulmonary vascular lesions and the DACA and cystic medial necrosis documented in the right coronary artery. Angiomatoid conversion of pulmonary arterioles may induce hypoxia or altered pulmonary blood flow which may, in turn, produce pathologic changes in coronary arterial walls. The absence of amphetamines in the toxicologic analyses lessens the possibility that they were implicated in the pathogenesis of the DACA. The possibility also remains that the patient had a forme fruste of Marfan's syndrome and the association with drug abuse and pulmonary vascular lesions was fortuitous.

Summary

A dissecting aneurysm of the coronary artery (DACA) associated with cystic medial necrosis was discovered during the necropsy of a 32-year-old black man with evidence of intravenous drug abuse. Microscopic examination of the lungs revealed striking angiomatoid conversion of pulmonary arterioles. The cause of death and the possible relationship between the pulmonary vascular lesions and the DACA are discussed.

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