

Acute INFLAMMATIONS of ARTERIES

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INTRODUCTION

INFLAMMATIONS of arteries are of especial importance largely because of the location and distribution of the arteries affected. Although it is not necessary in this discussion to determine whether arteriosclerosis is basically inflammatory or degenerative, yet it may be profitable to draw attention to the fact that its clinical importance is determined by what special arteries are diseased. For example, arteriosclerosis may be profound in the aorta, with little to show for it clinically, unless some complication occurs such as dissecting aneurysm or occlusion of peripheral arteries by lodgment of fragments of plaques or of bits of the thrombi which may form over atheromatous ulcers. In contrast, arteriosclerosis of peripheral arteries may give clear cut clinical manifestations. Affecting the coronary arteries there may be myocardial insufficiency and the sclerosis may furnish the basis for occlusion by thrombosis. Cerebral arteriosclerosis may lead to mental deterioration and the sclerotic cerebral artery is the one which ruptures to produce one of the forms of apoplexy. Sclerosis of arteries to the extremities may, when a thrombus forms, lead to ischemic necrosis, for example of the lower leg and foot when the popliteal artery is occluded. Parenthetically, it should be said that the term ischemic necrosis indicates simple necrosis, sometimes referred to as "dry gangrene." True gangrene ensues when the necrosis is complicated by invasion of saprophytes.

Similarly some of the disturbances due to acute arteritis depend on the artery or arteries affected, whether they be due to the physiological effects on the artery, as for example spasm, the extension of inflammation into surrounding structures, rupture of the diseased artery and consequent hemorrhage, or infarction as the result of accompanying thrombosis.

ETIOLOGY

For the most part, arteries become inflamed because of extension from neighboring foci or regions of inflammation and sometimes by deposit of pathogenic bacteria or other organisms in their walls. Arteries may be inflamed as part of certain infectious diseases, some of which, such as influenza, are of viral origin. The cause being known, these sorts of arteritis may be called secondary. In contrast, there are varieties of arteritis, either local or disseminated, in which the cause is not clearly defined, and Karsner has designated them as primary arteritis. Naturally, as time goes on, the cause of some or all of the latter will be identified and the term primary is thus provisional.

MORPHOLOGY

Sometimes, the disease in the arteries can be seen by the naked eye; this will be described in connection with the special varieties. Microscopically, several forms can be identified, as pointed out by Karsner. They will be described in accord with the following table, but it must be remembered that there may be a certain progress from one form to another and that frequently several of the forms are present at the same time. Special varieties of arteritis will be discussed subsequently.

Acute Arteritis

Alterative (degenerative)
Necrotizing
Exudative
Vegetative (thrombo-arteritis)
Proliferative
Organizing

Acute Alterative or Degenerative Arteritis represents for the most part just a degeneration of some sort in the arterial

wall. Such a condition was first described in 1869, by Hayem, in acute infectious diseases. Edema is the principal change, usually in the media. This may be within the muscle cells or between them and also in the subendothelial connective tissues in arteries large enough to have this layer. The lining endothelial cells may also be swollen presumably because of intracellular accumulation of water. Another change in the media is mucoid degeneration. This is common in the aorta and the mucoid of acute arteritis is evidently the same kind of connective tissue mucoid. Degeneration of elastica is indicated by swelling, splitting and fragmentation. That this degeneration is of inflammatory nature is indicated by comparison of large numbers of specimens which show progression to exudation.

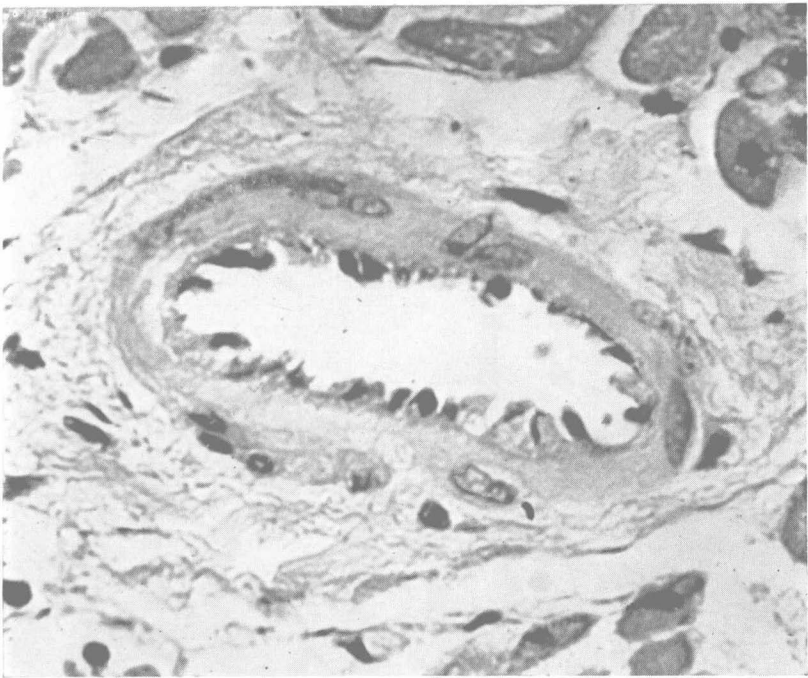


Fig. 1. Acute degenerative arteritis. This small artery in the myocardium is somewhat dilated and shows swelling of the lining endothelium. At one pole there is a vacuole indicative of edema in the media. In part of the media the nuclei have almost entirely disappeared. X 700. (Courtesy of Annals of Internal Medicine.)

In man, this condition occurs in a variety of acute infectious diseases, in rheumatic fever, polyarteritis nodosa, and as a consequence of poisoning such as by arsphenamine. Karsner quotes several authors as having shown it experimentally in hyperergic conditions, as a sequel of partial denervation, as the result of repeated grafting of adrenal tissues and as a result of prolonged life in compressed air.

Acute Necrotizing Arteritis. Although the terms necrosing and necrotizing are used interchangeably, we prefer the latter. Usually necrosis begins in the media and may extend to the intima and adventitia. The lesion affects a segment of the artery and may either extend around the entire circumference or be present in only one part of the wall. The muscle cells fuse and

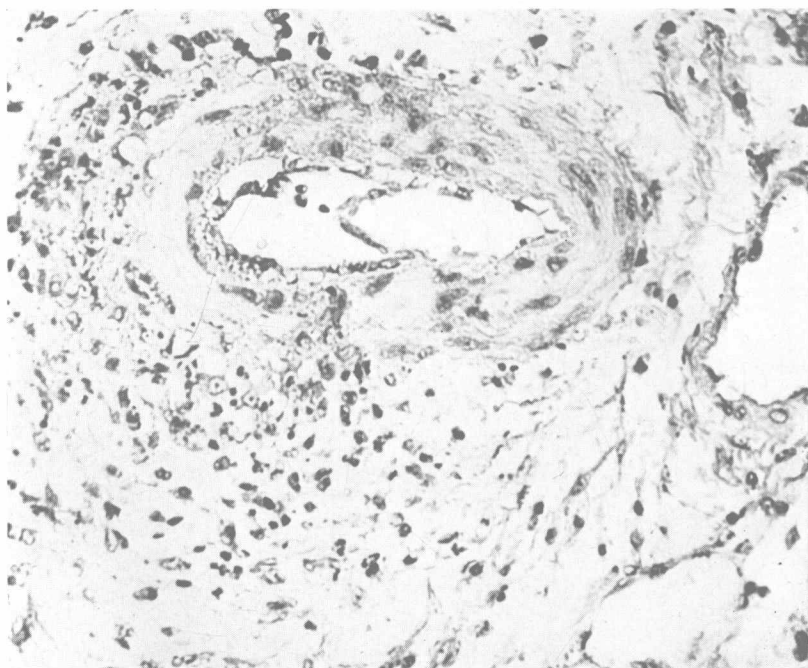


Fig. 2. Acute degenerative arteritis. The edema of media is seen at one pole but the swelling of endothelium is not striking. In this instance exudation has already begun, as illustrated by the mononuclear and polymorphonuclear cells around the artery with slight penetration into the adventitia. X 273.

become hyalinized and the nuclei disappear. Sometimes fragmentation and condensation of nuclei occur and in some instances the necrosis is granular rather than hyaline. The necrosis extends to intima and adventitia but is rarely primary in either of these coats. Cellular exudation of some degree is constant. In some cases, lymphocytes, plasma cells and large mononuclears are the only cells, in others they predominate and in others polymorphonuclear cells constitute most of the exudate.

In man, acute necrotizing arteritis is occasionally found in infectious diseases, especially when there is septicemia. It is seen sometimes in the florid cycle of rheumatic fever, in erythema nodosum, occasionally in various forms of typhus fever,

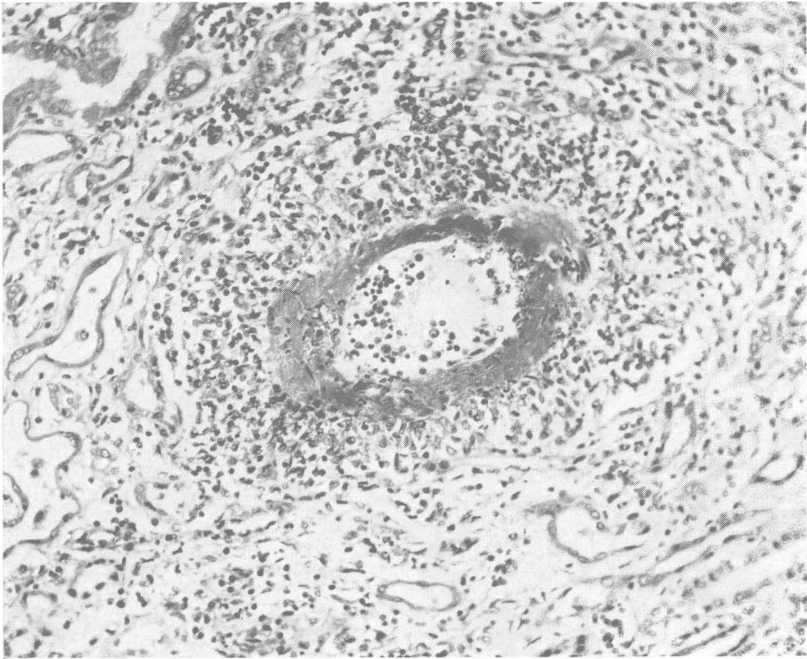


Fig. 3. Acute necrotizing arteritis. The wall of this artery is entirely replaced by a necrotic hyaline mass. Exudation is fairly extensive in the adventitia and surrounding tissue. This was found in the kidney without known cause. X 177. (Courtesy of *Annals of Internal Medicine*.)

other changes in the arteries, such as degeneration, necrosis and proliferation of cells, especially those of the subendothelial portion of the intima. This lesion occurs in various forms of primary arteritis and also in secondary arteritis, particularly when the arteritis is due to extension from a focus of suppuration. The experimental features will be discussed under the heading of suppurative arteritis.

Acute Vegetative or Thrombo-arteritis. Various authors have pointed to the fact that the thrombosis in these cases resembles that seen on cardiac valves, more particularly in the sort of thrombosis with little cellular infiltration such as occurs in acute rheumatic valvulitis. Naturally it cannot always be determined whether the thrombi are actually emboli from

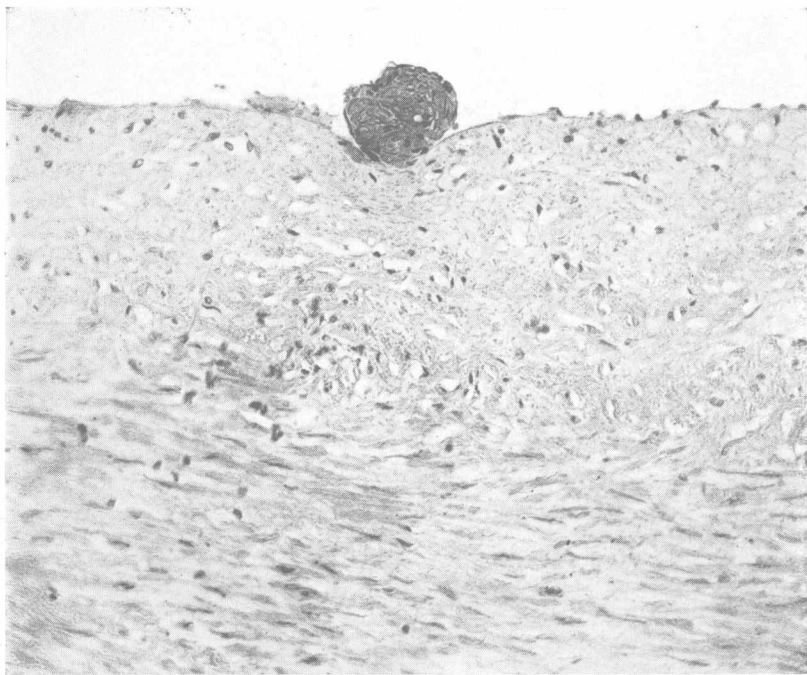


Fig. 6. Acute vegetative arteritis. Section of a large coronary artery in a patient with rheumatic fever. The thrombus is attached to the intima and resembles closely the verrucae which occur on cardiac valves. Inflammation is present in the depths of the slightly thickened intima but is not conspicuous. X 206. (Courtesy of *Annals of Internal Medicine*.)

Price and Herbut reported similar lesions in a case of trichinosis.

Acute Exudative Arteritis. As indicated above, exudation commonly accompanies acute necrotizing arteritis. Nevertheless there are varieties in which exudation is the conspicuous feature. This may be predominantly a fluid exudate, although that is rare. It is to be remembered that fluid exudates are in the form of an edema which is identified as inflammatory because of presence of fibrin. More frequent is cellular exudation which may in some instances be accompanied by the deposit of fibrin. As a rule the cells are in part mononuclear cells and in part polymorphonuclears. In some forms, such as polyarteritis nodosa, to be described subsequently, eosinophilic leucocytes are prominent in the picture. Acute exudative arteritis shows various

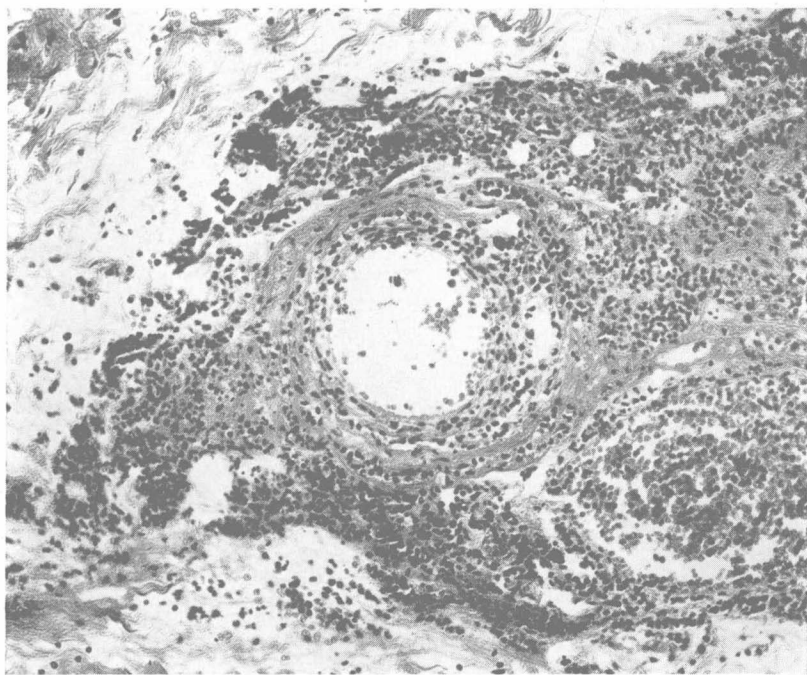


Fig. 5. Acute exudative arteritis. This was taken from the skin of a patient with erythema induratum. Extensive perivascular exudate, largely mononuclear, surrounds a small artery and is evident in media and intima. X 177.

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cardiac valves or whether the thrombosis is primary in the artery. Nevertheless, cases occur in which there is no disease of the heart and it is fair to assume that the thrombus forms in the situation where it is observed. Ordinarily these thrombi are comparatively free of cells but in some instances the cellular component may be considerable. When occurring in small arteries the thrombi may actually occlude the artery but even in small arteries the thrombosis may be non-occlusive and attached to only part of the lining. In the larger arteries it is not rare to find the thrombi situated on the wall but of relatively small size. In either event the question arises as to how much disease of the arterial wall is necessary before thrombosis occurs. This is hard to answer because in some instances the dis-

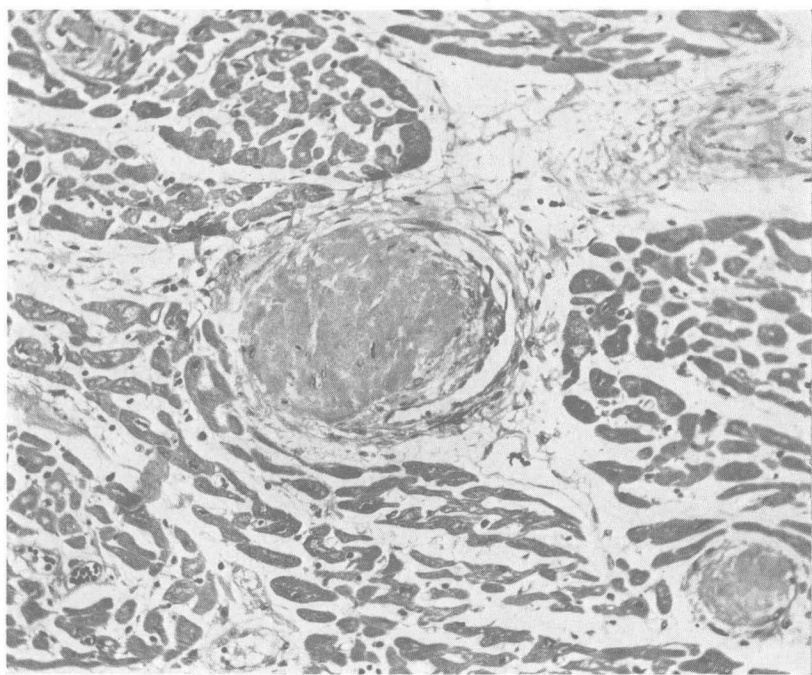


Fig. 7. Acute thrombo-arteritis. This affects a small artery in the myocardium of a patient with erythema induratum. The inflammatory component is exceedingly slight but the vessel is almost occluded by a thrombus made up largely of fibrin. X 177.

ease of the arterial wall may be so slight as not to be demonstrable morphologically. In most cases, however, there is some disease of the arterial wall which may vary from a simple degeneration all the way through to the most profound type of exudative or necrotizing arteritis. The lesion is found occasionally in rheumatic fever and it accompanies various other forms of arteritis. In some instances it would appear that a thromboarteritis may occur as a primary event. It is seen in acute infections of various sorts and occasionally is observed in the small arteries in typhus fever and other conditions.

Acute Proliferative Arteritis. In this condition the cells of the intima, more especially those of the subintimal connective tissue, show considerable multiplication. Ordinarily the fibro-

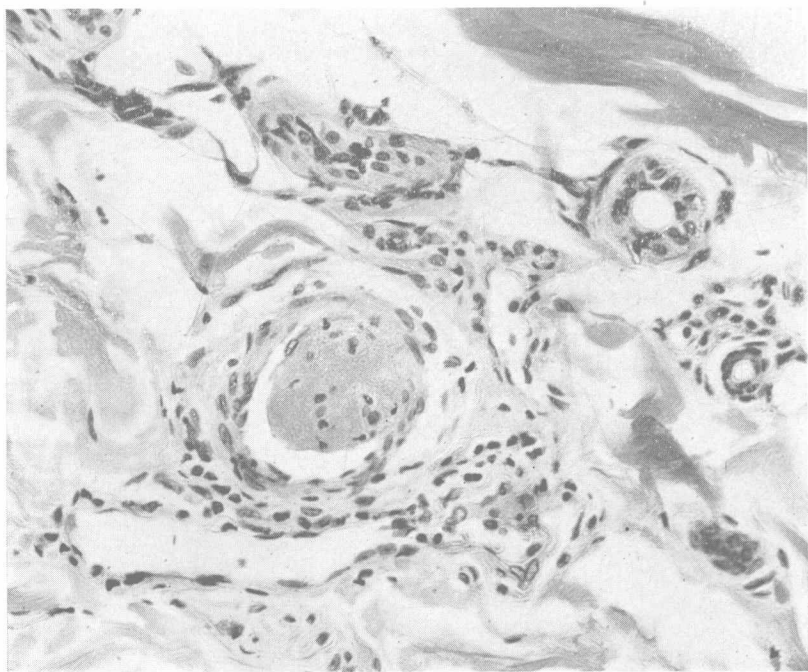


Fig. 8. Acute thrombo-arteritis. This is a small cutaneous artery from a patient with typhus fever. The cells of the exudate around the artery are principally mononuclears. The thrombus is made up largely of fibrin but contains a few mononuclear cells. X 285.

blasts are loosely arranged and may be infiltrated with cells of exudate. The proliferation leads to deformity of the lumen of the artery and may actually become occlusive. Not infrequently the media shows some cellular infiltration as is true sometimes of the adventitia. Curiously enough, thrombosis is not frequent in this particular form of arteritis. The arteritis may be entirely localized, as for example, to the appendix, to the gall bladder or to the kidney. In these cases the cause cannot be determined. It occurs occasionally in more widespread distribution in rheumatic fever and in polyarteritis nodosa. It is also said to occur in cases of acute syphilitic disease of the arteries. It has been reported as occurring in ergotism and experimentally as the result of repeated adrenal grafting. In those

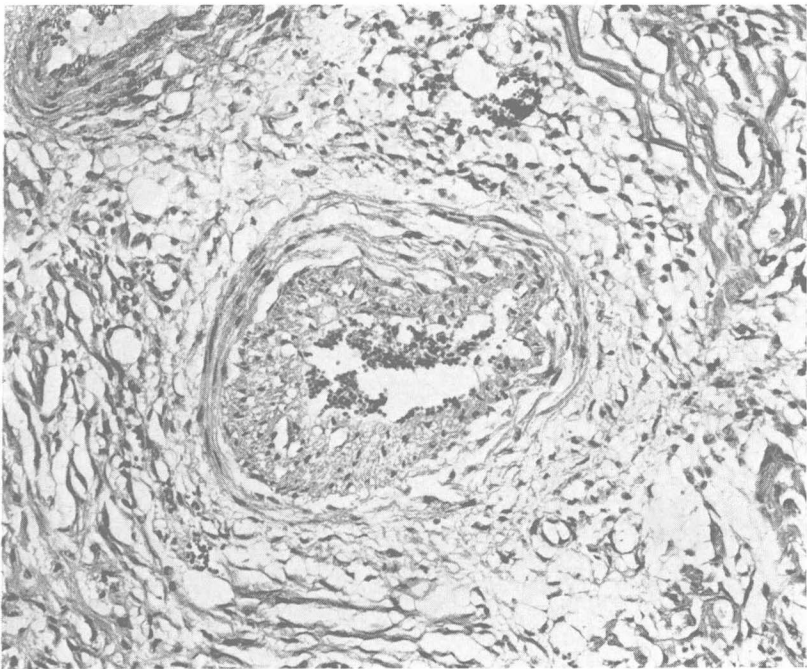


Fig. 9. Acute proliferative arteritis. This is a small myocardial artery in a young patient with rheumatic fever. The intima shows considerable proliferation of cells which are probably fibroblasts. There is slight edema of the media and of the surrounding tissue. Exudation is inconspicuous. X 158.