

# Pathology of Ruptured Saccular Aneurysms of Cerebral Arteries Causing Sudden Death

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## ABSTRACT

Fibrinoid necrosis of arterial walls, apparently of a primary nature, occurs quite frequently in the saccular aneurysms of cerebral arteries. A careful analysis of 25 cases of ruptured saccular aneurysms of cerebral arteries causing sudden death was made. All cases selected represented only the ones who had died instantly in public or whose instant death could be verified, in order to eliminate possible secondary necrosis. In 18 cases, genuine fibrinoid necrosis was observed involving all or parts of the aneurysmal walls. This furnishes an interesting speculation as to the pathogenesis of rupture in such aneurysms. An additional attempt is made during the course of this study on the pathogenesis of saccular aneurysm itself to reveal abnormalities in the elastic fibers to be the primary cause for such aneurysm formation. Other possible mechanisms are also discussed.

Upon examining a series of saccular aneurysms of cerebral arteries, the author was impressed by the frequent presence of fibrinoid necrosis in the aneurysmal walls, particularly when there was a rupture. The necrosis was not only observed in the vicinity of the rupture but also in the areas where structural integrity was maintained. Notwithstanding the pros and cons on the pathogenesis of

saccular aneurysms of cerebral arteries, the initial purpose of this study was to clarify the relationship between fibrinoid necrosis and the pathogenesis of rupture in so-called "berry aneurysm" of cerebral arteries.

Although the congenital origin of this type of aneurysm appears firmly established, there have been many who emphasize the importance of degenerative vascular changes playing a greater role in the formation of cerebral aneurysms. (Mitchell et al., 1943; Richardson et al., 1941; Tuthill, 1933; Walker et al., 1954) During the course of the present study, the author was also impressed by the remarkably high association of degenerative changes in the vessels. An additional attempt is made to identify the nature of these degenerative vascular changes and their possible role on the pathogenesis of such aneurysms.

## MATERIAL

The materials for this study come from the autopsy files of the coroner's office of the Cuyahoga County, Cleveland, Ohio, U.S.A. Since fibrinoid changes in the vessel walls may be observed in a number of conditions as a secondary phenomenon, such as in an area of severe inflammation, the cases selected represent only the ones who died instantly in public or whose instant death could be verified, in order to eliminate the possibility of marked inflammatory reaction in areas of rupture. The cause of death in all was attributed to ruptured cerebral aneurysm with resultant diffuse subarachnoid hemorrhage. The series consists of 25 cases; in all, one or more slides were

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available, and in 11 cases, serial sections of the aneurysms were studied. The sections were stained with hematoxylin and eosin, Mallory's azan and Verhoff's elastic stain. The average age of the patient at the time of death was 40.64 years, with the extremes of 8 and 53 years. Thirteen patients were male and twelve female. The majority (19) were white. In one case there were 3 aneurysms in different locations, and in one, there were two.

### ***PATHOLOGY***

#### *Location of aneurysms*

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Anterior cerebral artery .....	9
Middle cerebral artery .....	5
Posterior cerebral artery .....	3
Basilar artery .....	3
Anterior communicating artery .....	2
Not certain .....	7

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With the exception of 2 fusiform aneurysms in the basilar artery, the aneurysms were of the saccular type grossly. The size of the aneurysms averaged approximately 1.0 cm. in diameter.

In 18 cases, definite fibrinoid necrosis in the aneurysmal walls was observed. The necrosis was invariably associated with rupture, but was also frequently observed in intact areas. Often the entire thickness of the fibrous arterial wall was involved by the process, but in other areas the necrosis was noted haphazardly scattered within the arterial walls. The degeneration was observed not only in the hyalinized connective tissue but also in the muscle fibers. Azan stain brought out very sharply large droplet-like precipitations of fibrinoid material. Although in a few cases there was very minimal inflammatory cellular reaction near the ruptured area, in the majority the necrosis was not associated with any form of inflammatory reaction.

The much-discussed muscular defect in the aneurysmal sacs were observed in all cases. More significantly, however, there was a moderate to marked degree of arteriosclerosis in almost all cases. This is significant, because other portions of the cerebral arteries in most cases did not show any appreciable degree of intimal proliferation. The arteriosclerosis was essentially of two varieties. The one most com-

monly observed was of the atheroma type with deposition of large lipid-laden macrophages and cholesterol slits, etc., predominating. The other was characterized by proliferation of young fibroblasts and mesenchymal cells. Nodular projections of a fibrous tissue mass were a very common finding.

The most significant of all the author's observations in this connection was that of the elastic fibers. All aneurysms studied showed one or another of the abnormalities in the appearance and arrangement of elastic membranes. The changes are characterized by marked thickening, homogeneous degeneration, splitting and fragmentation. The abrupt absence of the elastic membrane in the aneurysmal sac was also very common. In some, the aneurysmal wall was completely devoid of elastic tissue. Often a fibroblastic form of intimal thickening was seen over these degenerated elastic membranes. Sometimes a stretched-out elastic membrane appears to hug atheromata or fibrous plaques filling the saccular out-pouches.

### ***COMMENT***

Identification of fibrinoid necrosis in the arterial walls of saccular aneurysms offers an interesting speculation as to the final events or the mechanisms involved in aneurysmal rupture. The necrosis observed quite convincingly appears to be a primary phenomenon rather than a secondary one. Although this phenomenon cannot be the sole explanation of rupture in such aneurysms, at least in many such might be the case. Fibrinoid necrosis of arterioles in hypertensive petechial hemorrhages in the brain is often thought to be the responsible factor also. Although figures vary from one another, hypertension is a quite common association in saccular aneurysms. None of our cases furnished recorded evidence of hypertension, but some showed myocardial hypertrophy. Whether the fibrinoid necrosis seen in this study represents the type of necrosis seen in arterioles of hypertensive patients or not can not be stated with certainty.

It should be emphasized, however, that there must be other mechanisms also playing a role in the final events. Emphasizing anomalies of the circle of Willis, Turner (1946) demonstrated the alteration of hemodynamics in such anomalous vessels to be the cause of

rupture. Reuterwall(1923) mentioned "a small rent in a diseased wall of a vessel" to be the cause. In any event however, the fibrinoid necrosis associated with the aneurysms studied appears to be a genuine one, and this certainly must be playing a greater role in the causation of rupture. Mitchell and Angrist (1943) mentioned a necrosis in the neck of the aneurysm but did not state the nature or the significance except to speculate on some unknown etiologic factor.

As for the etiology of saccular aneurysm itself, there have been numerous investigations in this connection. Congenital defects in the muscular coats of cerebral arteries have long been recognized, and some have placed great significance on them for the development of such aneurysms (Forbus, 1930). However, considering the fact that such defects can be found in the cerebral vessels and other vessels in the majority of normal people (Glynn, 1940), many have attempted to identify other mechanisms for the formation of aneurysms.

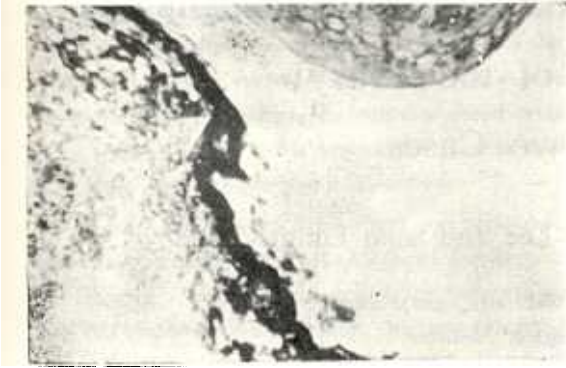
It is true, however, that the prevailing opinion among workers in this field tends to favor the congenital origin of cerebral aneurysms. Riggs and Rupp (1943), for instance, pointed out the frequent association of anomalies of the circle of Willis and sought the cause in the altered intravascular hemodynamics due to such anomalies. Walker and Allegre (1954) emphasized the importance of atherosclerosis, on the other hand, and placed greater significance on this primary vascular disease. Tuthill (1933) also emphasized atherosclerosis as the cause, but he denied the presence of muscular defects in such aneurysms.

Musclar defects probably represent a genuine defect, and there is no doubt that they play a part in causing aneurysms; but considering that these aneurysms are comparatively rare and that such defects are a very common finding in normal individuals, there must be other factors precipitating aneurysm formation. The importance of elastic tissue has been pointed out by a number of other workers (Glynn, 1940; Richardson et al., 1941; Carmichael, 1950).

Observations in this study strongly favor the opinion that the pathogenesis of cerebral aneurysms is related to a primary alteration of the elastic tissues in the circle of Willis and that atherosclerosis probably is a secondary phenomenon. Other factors also undoubtedly play a part, but the elastic tissue alteration must be the ultimate cause.

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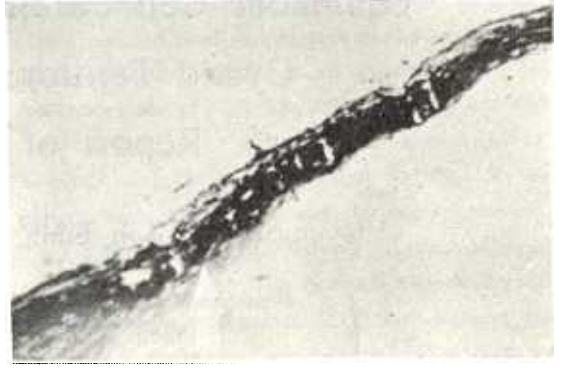
**Fig. 1.** Case No. M-12,142. 51-year-old white male who died instantly while shopping. Aneurysm was found in the right middle cerebral artery. The picture shows homogeneous degeneration of aneurysmal wall involving the entire thickness at this area. Azan.  $\times 100$ .



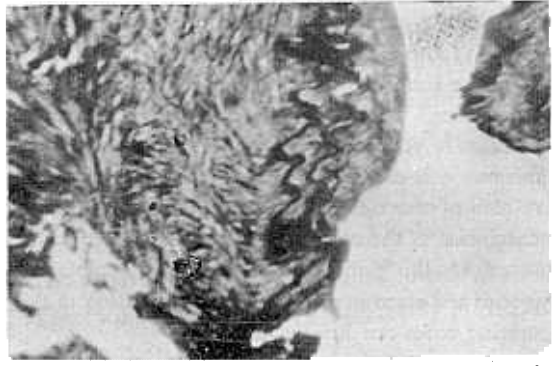
**Fig. 3.** Case No. M-90,77. 46-year-old white female who collapsed at wheel. Aneurysm was found in the right anterior cerebral artery. Note large droplet-like precipitation of fibrinoid substances in fragmented aneurysmal wall. Azan.  $\times 430$ .



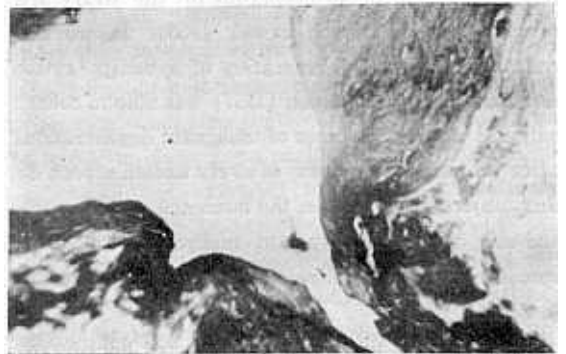
**Fig. 5.** Case No. M-6,792. 36-year-old white male who collapsed in his basement and died instantly. Note marked atherosclerotic plaque over degenerated elastic membrane. EVG  $\times 100$ .



**Fig. 2.** Case No. M-10,687. 46-year-old white male who collapsed in the bath room and died instantly. Aneurysm was found in the anterior communicating artery. The picture shows fibrinoid necrosis of aneurysmal wall. Azan.  $\times 100$ .



**Fig. 4.** Case No. M-9,654. 38-year-old white male who collapsed at work and died instantly. Site of aneurysm not clearly stated. Note irregular splitting of elastic membranes over which proliferation of intima is evident. EVG  $\times 100$ .



**Fig. 6.** Case No. M-8,150. 53-year-old colored male who became suddenly ill at home and died very acutely. Aneurysm was found in the right anterior cerebral artery. Note marked atheromatous plaque devoid of elastic membrane.