An Approach To The Etiology, Diagnosis And Management Of Peripheral Arteriosclerosis (Arteriosclerosis Obliterans)

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ETIOLOGY

Articles on arteriosclerosis, especially on the relationship between the disease and diet, are appearing in increasing numbers, both in lay and professional literature. No definite etiology has yet been established and hence no specific therapy can be prescribed, dietary or otherwise. The disease is currently believed to be a metabolic disorder, a disturbance of lipid metabolism, which by some obscure means leads to accumulation of cholesterol in the lining of the arteries. The latter process is only one phase of arteriosclerosis; namely, atherosclerosis. Gofman¹,² says that atheroma formation has two major features: 1) a definite focal character of the lesions and 2) an abnormality in the serum lipids associated with the development of the disease. He states further that the most plausible concept of the pathogenesis of arteriosclerosis is that certain circulating lipids are deposited and retained in susceptible focal areas.

The role of obesity in arteriosclerosis has been discussed by many writers, including Dublin and Marks³, Gofman¹, and Gofman and Jones². However Keys⁴ is most emphatically positive about the direct relationship, neither including nor excluding the aging factor. He has shown statistically that there is an increasing incidence of fatal atherosclerosis in people who are overweight and that during periods of semi-starvation, deaths from vascular disease declines. In autopsy cases, the severity of atherosclerotic vascular degeneration was directly related to the degree of fatness of the whole body. He believes that though atherosclerosis has not been proved to be due to eating fat or cholesterol and probably is not due only to dietary factors, yet substantial control of atherogenesis may be achieved by avoiding excessive calorie intake and obesity and by limiting the fat content of the diet so that the extractable fats provide almost 25 to 30 per cent of the total calories.

Steiner, Kendall and Mathers⁴ found serum cholesterol and serum lipid-phosphorus values elevated in patients with coronary artery disease as compared with a control group of apparently healthy normals. The increase in serum lipid-phosphorus was not proportional to the increase in serum cholesterol, resulting in an increase in the serum cholesterol-lipid-phosphorus molar ratio, which is of possible significance in atherogenesis.

Katz⁷ says the basic tenet is that altered lipid-cholesterol metabolism is necessary for atherosclerosis to develop. Senescent changes in the artery wall are not considered of prime etiological importance. But Lansing⁸ feels that the altered lipid-cholesterol metabolism concept is not entirely satisfactory. It does not explain the fact that some plaques are entirely fibrous and do not contain cholesterol. Neither does it explain the focal nature of arteriosclerotic lesions, the greater severity of lesions in the abdominal than the thoracic aorta, or the relative resistance of some arteries, such as the pulmonary, to plaque formation, and finally, it does not explain the connection between cholesterol metabolism and luetic aortitis or mitral stenosis, both of which are characteristically associated with severe arteriosclerosis. Lansing has shown quite conclusively that calci-*Physician-in-Charge, Medical Clinic No. 2
fication of senescent tissue in the media of human arteries occurs with or without atherosclerosis and that elastic tissue changes begin as early as the third decade. When these changes and atheromatosis coexist, cholesterol accumulates in the intimal plaques after the underlying media has undergone elastic tissue calcification.

It would seem logical, then, to say that arteriosclerosis is a dual process: 1) aging of the arterial wall, establishing a substrate for 2) penetration and accumulation of cholesterol. It is possible that without such changes in the artery wall, whatever the factor or factors that condition the age change, cholesterol will not accumulate in the lining of the arteries. We cannot but agree with Moschowitz when he says, "Arteriosclerosis is a compensatory reaction to normal arterial pressure which we cannot escape. Hence, it is the inevitable destiny of all animals (humans included) that possess a vascular system and live long enough."

**DIAGNOSIS**

There should be little trouble in the diagnosis of peripheral arteriosclerosis (arteriosclerosis obliterans) if an accurate history is obtainable. We know of no other condition that produces calf-muscle pain in one or both legs which comes on only with walking or other leg strain and disappears with rest. If similar pain involves the muscles of the thigh and hip, either alone or in addition to leg muscle pain, then segmental occlusion of larger arteries, femoral or iliac, must be thought of, either alone or together with occlusive arterial disease more distally.

What can be seen and felt on examination of the extremities is of next importance. With practice one becomes proficient at estimating the degree of arterial occlusion from palpation of the major arteries of the extremities. This must always be judged in connection with the history and other findings, since one or more of the pulses in the feet or at the wrists may be absent in a fair percentage of normal people. For the upper extremity, the Allen test will determine whether there is impairment of flow in radial or ulnar arteries. Even with palpable pulses in major foot or wrist arteries, smaller vessels may be narrowed or occluded and permit actual gangrene or ulceration of the part supplied by such distal branches. Skin color often gives helpful information. Increased blanching on elevation of the legs and red or bluish discoloration on dependency are characteristic of leg artery insufficiency. Frequently there is decreased temperature of the skin of the extremities, slow nail growth or transverse ridges on the nails.

Laboratory aids are often used to help in diagnosis. X-rays for blood vessel detail may show calcification, at least helping to exclude thromboangitis obliterans in a borderline case. It should always be remembered that calcification of the artery wall is no indication of the degree of arterial impairment and that failure to visualize calcium does not rule out arteriosclerosis or arterial insufficiency. In a recent series of 54 patients with arteriosclerosis obliterans, 32 had x-rays of the legs. Twenty-three were positive, 9 negative for calcium deposits in the artery walls. The age range for the positive cases was from 49 to 72 years and for the negative ones from 46 to 62 years. There was no direct relationship between the degree of calcification or its absence and the intensity of symptoms, nor was the degree of calcification related to age.

Aortograms and femoral arteriograms are being done quite routinely now to de-
tect the cases of segmental occlusion in the larger arteries of the lower extremities. The histamine-fluorescin test at times aids in deciding the site for amputation. Oscillometry is often done but seems of little value over palpation for pulses. Vasodilator tests are used regularly and give much help. In our experience, Priscoline intravenously (50 mgms.) has proven very satisfactory in most cases. When clinical findings are not corroborated by the results of dermatherm study with this drug, the temperature readings are repeated with lumbar sympathetic block or spinal anesthesia. These studies are carried out under constant temperature conditions, of course.

MANAGEMENT

Regardless of the controversies as to etiology, the fact remains that with the increasing number of men and women living to and beyond three score years and ten, physicians of today are faced with arteriosclerosis as the leading disease of the age. It naturally follows that there will also be more patients with arteriosclerosis of the extremities. Some will have symptoms and come for help. As already stated, no specific treatment is known. We cannot be as optimistic as those who feel that diet alone or a new vasodilator drug is the answer. But neither can we agree that little or nothing can be done once the disease blocks or narrows the lumen of the peripheral arteries. There must be a mid-way program, not specific therapy, but a plan of management which will effect an increased blood supply to an extremity with partially obliterated arteries.

To begin with, as Duryee says, each case presents a different set of problems which must be managed from the standpoint of the patient's general condition and from proper evaluation of the actual vascular changes in the extremities. Certainly no one vasodilator drug will be effective in every case. Neither will sympathectomy be regularly advised or helpful. The plan of management should be for the patient as a whole, not just for the offending extremity.

It is inescapable that there will be some patients with sudden occlusion of one or more larger leg arteries with resultant severe pain, gangrene or other complication requiring major surgery, such as amputation. Thrombosis is more likely to occur in arteries with atheromata and calcification. It is the dreaded and important complication in arteriosclerosis. Fortunately, however, most patients with peripheral arteriosclerosis come to us when still ambulatory, with no or only minor complications, such as small ulcers or minimal gangrene. Even these require careful cooperation of internist and surgeon, so that the unfortunate few requiring necessary major surgery, may have it carried out without prolonged suffering. But the large majority will respond to conservative measures, for which we make a plea. These include the following:

1. Teaching patient and family the nature of his disease. Then he and they will appreciate the importance of the time element for improvement, which is always slow. It will mean for patience and better cooperation.

2. Training the patient and his family in good extremity care. Daily foot baths followed by cautious drying and application of foot powders lessen the chance for fungus infection. If the skin is dry or scaly, lanolin or other skin oil may be applied. Corns, callouses and other foot disorders should never be self-treated. Trauma of any type should be avoided. Soft, closely woven stockings and well-fitting shoes will tend to
reduce the danger of irritation and will keep the skin warm. In colder weather, whole body warmth is important, meaning long underwear, wool socks and other appropriate clothing.

3. New habits of activity. If there is much pain, or there are small ulcers or gangrene, hospitalization or at least rest at home for two or three weeks is often necessary. When again ambulatory the patient should know that some walking or other activity is actually helpful in promoting collateral circulation. But he must learn to walk slowly and only short distances, keeping activity just under the point of pain. Buerg er exercises and contrast bath followed by gentle massage to the legs are also recommended, not only for the slight help this may give but also to bolster the patient’s morale; he is doing something for himself.

4. Diet. Though there is no conclusive evidence that excess fats are a definite etiological or aggravating factor in arteriosclerosis, it is important to control weight. The best way to correct obesity is through a low fat diet and this, then, also controls the possible damage from excess lipids. But this does not mean the extremely low fat diets often prescribed which, in our opinion, are as crippling as the rice diet of Kempner. We cannot agree with Keys that butter, cream, oleomargarine, salad oil, mayonnaise, fried foods, gravy, ice cream, chocolate and pastry containing shortening should be prohibited. The diet should be low in fat, but can still be palatable, attractive and balanced.

5. Preventing vasoconstriction. This means absolutely no tobacco and no vasoconstrictor type of drug. It necessitates avoiding chilling, not only of the extremities but also the body generally. Finally it requires control of nervous tension, anxiety, fear, anger or other emotional disturbances which are always aggravating factors.

6. Promoting vasodilation. Alcohol is without doubt the best vasodilator. But it must be prescribed with caution, as a medicine only, and preferably not more than an ounce of whiskey, or its equivalent in other alcoholic beverages, three times daily. If dermatherm study with intravenous Priscoline reveals residual arterial elasticity with a skin temperature rise of two or more degrees centigrade, this or other vasodilator drugs by mouth have some value. A new preparation, Arlidin, is reported to be the most satisfactory for dilatation of the deeper muscle arteries. Intra-arterial histamine has been lauded as a limb-saver by Mufson, but our limited experience with this drug has not been encouraging. It carries with it the danger of local arterial thrombosis or thrombophlebitis. Sympathectomy in selected cases gives added warmth to the skin and is especially helpful in patients who suffer from coldness and superficial ulceration. It should not be expected to relieve pain.

7. The use of mechanical aids. Various types of physical and mechanical therapy have been used and at times are helpful. Buerg er exercises and massage have already been mentioned. Dry and moist heat must be used cautiously to avoid burns. Tissue with impaired circulation does not tolerate either extreme of temperature well. The safest measure is a light warm blanket wrapped around the extremity and covered with a loose cotton boot, to retain natural body heat. A heat cradle with the thermostat at 92 to 96 degrees Fahrenheit may be placed over the involved extremity. Reflex heat from diathermy over the sacral area is often comforting. If there are no
open lesions, Sitz baths may be used, 20 to 30 minutes, with the temperature of the water at 94 to 100 degrees Fahrenheit. For open lesions, moist compresses of warm normal saline solution for an hour three times daily are soothing and cleansing. Any preparation that irritates or increases pain should be strictly avoided. Without doubt the oscillating bed is the most helpful mechanical device. Patients with quite severe pain have been known to fall asleep on these rocking beds when many other attempts at producing comfort have failed. We have seen little help from venous compression, alternate suction and pressure and other mechanical aids so often recommended by the manufacturers of expensive apparatus.

8. Adequate treatment of other associated diseases, such as diabetes, osteoarthritis, blood dyscrasia, diseases of the spinal cord, vitamin deficiency, endocrine dysfunction or orthopedic defects.

In the previously mentioned series of 54 patients followed from 2½ years to 5 years, 36 had conservative medical treatment only. None developed complications and only two failed to respond satisfactorily. Both of these refused to stop tobacco. Of 12 who had medical therapy and sympathectomy, three continued to have pain and other untoward symptoms. All three also continued smoking. The remaining six were quite advanced cases for whom sympathectomy was advised in the hope of avoiding amputation but later required radical surgery because of unbearable pain and advancing gangrene. Three required unilateral supracondylar amputation and are doing well with prosthesis. Two had bilateral amputation but are also comfortable now and continuing some gainful occupation from a wheelchair, three years postoperatively. The final patient who had a cerebrovascular accident and a coronary occlusion one year before the final admission, got no help from sympathectomy and expired of a second coronary occlusion following amputation of the involved leg.

Analysis of these three groups shows no significant difference in age range except that the six requiring amputation were 55 years or over. It was interesting, too, that the three patients who continued to have pain and other symptoms after sympathectomy were 46, 48 and 49 years of age.

SUMMARY

There is still no definitely known etiology for arteriosclerosis but it seems reasonable to say that it is the result of a dual process; namely, 1) aging of the arterial wall, establishing a substrate for 2) penetration and accumulation of cholesterol from a disturbance in lipid metabolism. The disease is doubtless present in every human who lives long enough but produces symptoms only in those who do not develop adequate collateral circulation to compensate for occlusion of the original vessels. There will always be some with sudden occlusion of a major artery, with ulceration or gangrene following trauma, or with segmental occlusion of a femoral or larger vessel, in whom the primary treatment will be surgical. But the majority of people with the disease will be ambulatory. For these a plea for conservative medical measures of therapy is made. Probably most important is the education of the patient and his family in an understanding of the nature of the disease so that there will be patience and cooperation. There are no miracle drugs, special diets, or gadgets that mean for quick recovery. There is only control, not cure.
BIBLIOGRAPHY