

Acute Coronary Occlusion In The Negro

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THE Negro race is susceptible to every disease that the white race acquires when he attempts to enjoy civilization. The incidence may vary depending upon many complicated immunologic factors.

Coronary artery disease is a metabolic disease. Coronary artery occlusion whether acute or the result of a marked narrowing of the coronary arteries represents a syndrome, the ischemia (the effect of the impaired blood supply to the heart) resulting in nutritional impairment of the heart muscle.

Coronary insufficiency occurs numerous times where cardiac insufficiency exists and can lead to necrosis of the heart muscles.

This is seen in aortic stenosis¹ shock, pulmonary embolism,² and from sudden hemorrhage. Organic changes need not be present in the coronary arteries to obtain myocardial infarction.

The condition of the cardiac muscle is graphically seen in the electrocardiogram which mirrors the circulation to the heart muscle and specifically the coronary blood flow. Significant changes in the electrocardiogram may be seen as a direct result of the anoxemia of the heart muscle. These changes may appear more often when coronary disease is present, but may be manifested in its absence.

The diagnosis of acute coronary artery occlusion is a clinical one and it is my impression, a very infrequent one in the Negro race.

The special cardiac clinic at the Mount Sinai Hospital in New York where I have been a research member for the past seven years has revealed fifteen cases during our studies. All of these cases had the serial electrocardiographic changes plus the typical course of an acute coronary artery obstruction.

This present year we have had two cases, one in a man with hypertension, age 35. We can no longer say that it is a rare disease among Negroes. I recently saw a case in a Negro woman, age 60,

who had hypertension and the coronary anginal syndrome who had radiation to her gall bladder which on x-ray visualized previously gall stones (cholelithiasis). She was found dead in bed two days later. The pain may be localized over the diseased gall bladder and it is almost impossible to make the correct diagnosis in these cases; usually the icterus is slight in acute coronary obstruction while, if the patient lives long enough, the jaundice deepens in acute cholecystitis. Necropsy reveals many errors by the best of clinicians. I have seen one other case in a Negro male. The experience at the Mount Sinai Hospital is about two cases a year. This hospital draws more Porto Ricans and relatively few American Negroes.

J. L. Hall, formerly of Provident Hospital, and now of Howard Medical College, Washington, D. C., stated to me personally in 1937 that he saw the disease on the increase and that the incidence of cases was about eight to ten a year at the hospital.

Bythewood of St. Louis General No. 2, Mo. states that he saw about twelve cases a year.

Garnes and Johnson of Harlem Hospital, New York, state that their evidence is about ten cases a year.

We must realize that the disease is definitely on the increase in the Negro race, and that it is not as rare as Farmer's³ experience would lead one to believe.

In congestive heart failure it is very difficult to diagnose an acute coronary artery occlusion. The changes of anoxemia of the heart muscle may give electrocardiographic patterns which are very misleading.

In Community Hospital we see the advanced stages of cardiac diseases, but clinically I have not seen a case of coronary artery occlusion.

Angina pectoris (pain in the chest) is a general term and must be differentiated as to etiology. Coronary artery angina pectoris is not seen commonly in the Negro. Dyspnea appears more often

where hypertension is present and the coronary "T" waves appear, coronary angina pectoris is probably present. Hypertension can cause an aortalgia and this can simulate anginal syndrome also. Coronary sclerosis is found at post mortem commonly in the Negro, but angina pectoris is not commonly seen ante mortem. I have seen numerous cases of definite angina pectoris in the Negro of coronary origin.

Coronary artery disease is not a disease of the intelligent alone. It is seen among poor people also. Some authors have stated that the intellectual criteria of the Negro is low and he does not register. This is categorically false. The poor uneducated white patient has coronary artery disease. Negro patients act and register identically as white patients where angina is seen.

Emphasis should be given to this disease which is elusively evincing itself in the Negro race and despite the want of laboratory facilities or even with the negative evidence of adequate laboratory facilities it remains a clinical diagnosis.

It is true that a cardiologist is in a better position to make the diagnosis, but it remains a fact that the otolaryngologist is hardly necessary to diagnose tonsillitis.

Dr. Dublin of the Metropolitan Life Insurance Co. states "that many cases are undiagnosed relative to coronary artery disease" is a liability against Negro doctors who handle most of the race patients in America.

Any physician who has ever seen a typical case of acute coronary arterial occlusion never forgets that patient.

This article is written to arouse an alertness in the Negro physician to the existence of coronary artery disease in the Negro race.

SUMMARY

1. Coronary artery disease is not rare in the Negro and is on the increase as to clinical diagnosis.
2. The diagnosis of coronary artery disease is a clinical diagnosis.
3. The course of the disease is the same as in other races.
4. The incidence in both races contrary to present views probably is the same.

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Mycotic Dermatitis from Straw Slippers

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SINCE 1937 I have observed three instances of dermatitis about the feet contracted from the wearing of straw slippers. The third instance is recent. The other two have been subject to recurrences in the spring and early summer although exposed footwear had been discarded. Unstained preparations in N/10 sodium hydroxide have revealed yeast cells in each case.

The lesion begins as a hard raised painless papule. When it occurs on the sole, it is found

usually in the search for a foreign body that is making walking difficult. The papule becomes a vesicle. Then the lesion becomes painful and is surrounded by a red, inflammatory areola. New lesions appear in this areola. If left undisturbed the vesicle becomes a pustule. Coalescence is the rule. The skin over the pustule breaks and sets free a thin, dirty gray pus in which the yeast is found. The base of the newly formed ulcer is red and painful, but I have not observed it to ex-