SUBENDOCARDIAL MYOCARDIAL INFARCT*

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THIS is a report of a case of extensive subendocardial myocardial infarction involving the interventricular septum and the anterior wall of the left ventricle. There was no coronary occlusion, and the electrocardiogram showed no evidence suggestive of a recent myocardial infarct. The rarity of this condition warrants a case report.

CASE REPORT

An 82 year old white female‡ was admitted for the first time after she had collapsed following the extraction of two teeth. Diabetes mellitus was diagnosed at that time, and the Wassermann and Kahn reactions were found to be positive (4 plus). After six days of treatment with insulin and diet the patient was discharged with the diabetes improved. About one year later she was re-admitted following an injury to the left foot complicated by dry gangrene of the fourth left toe. During her stay at home the patient had not taken any insulin, and she had followed her diet inadequately. The family history disclosed that one brother died at the age of 69 of coronary thrombosis. She was gravida VI, para II, with three premature births and one stillbirth, and two live children. On admission her chief complaints were pain and swelling of the left foot and some frequency in urination, urgency and incontinence without dysuria. Physical examination revealed a well-developed, well-nourished white woman, resting comfortably in bed. The temperature was 98.6° F., the pulse was 88 per minute, the rhythm was regular. Respirations were 20 per minute, arterial blood pressure was 168 mm. Hg systolic and 70 mm. diastolic. The heart was enlarged to the left, reaching the anterior axillary line in the fifth intercostal space. A high pitched systolic murmur was heard at the apex and the aortic second sound was louder than the pulmonic. The fourth toe of the left foot was bluish-black, and there was reddening and swelling of the dorsum of the foot. The pulse of the left dorsalis pedis artery was not palpable. The clinical diagnosis was gangrene of the left fourth toe and generalized arteriosclerosis. The hemoglobin was 10.4 gm., the erythrocyte count was 4,660,000 per cubic mm. and the leukocytes were 14,300 per cubic mm. with a normal differential count. The fasting blood sugar was 250 mg. per cent, and the non-protein nitrogen was 40 mg. per cent. Twenty-four hour catheterized urine specimen showed an acid reaction, specific gravity 1.013, sugar and tests for acetone were negative. The sediment showed three to five white cells. Roentgenogram of the left foot revealed a fracture to the base of the fifth metatarsus.

The patient was placed on a diabetic diet, received acetylsalicylic acid and sulfadiazine (gm. 1, every 4 hours). Local heat and moist application produced some local improvement.

The temperature ranged between 98.6° and 100° F., and her general condition was good for five days when the temperature rose to 101° F. without elevation of the respiratory or pulse rate. Twenty-four hours later, on the sixth night after admission, the patient suddenly became very restless, had an emesis and an involuntary bowel

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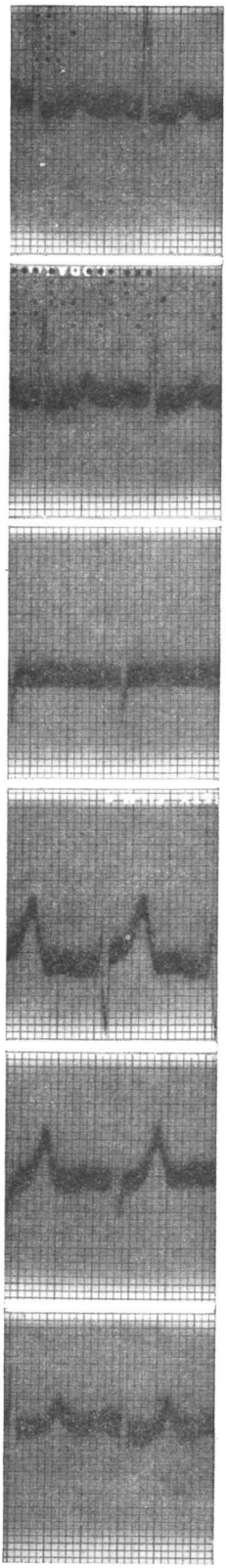

FIG. 1.

movement. She developed marked dyspnea, râles were heard over the lungs and mucus accumulated in her throat. The blood pressure at the beginning of this episode was 116 mm. Hg systolic and 60 mm. diastolic and slowly dropped to 100 mm. Hg systolic and 60 mm. diastolic. The pulse was of fairly good quality and regular, with a rate of 100 to 110. The patient vomited twice more. The impression was that this attack was caused by an acute coronary occlusion. The possibility of a pulmonary embolus was also considered. Oxygen, aminophyllin and morphine sulfate were administered, and the patient improved. Urine examination at that time revealed 4 plus sugar but no acetone; the vomitus showed a 4 plus occult blood. The electrocardiogram taken 17 hours after the beginning of the attack disclosed a definitely abnormal curve with left ventricular preponderance, and no evidence of a recent myocardial infarct. The patient was comfortable and in good condition for 36 hours following the attack, then suddenly she started to gasp for breath, became pale and

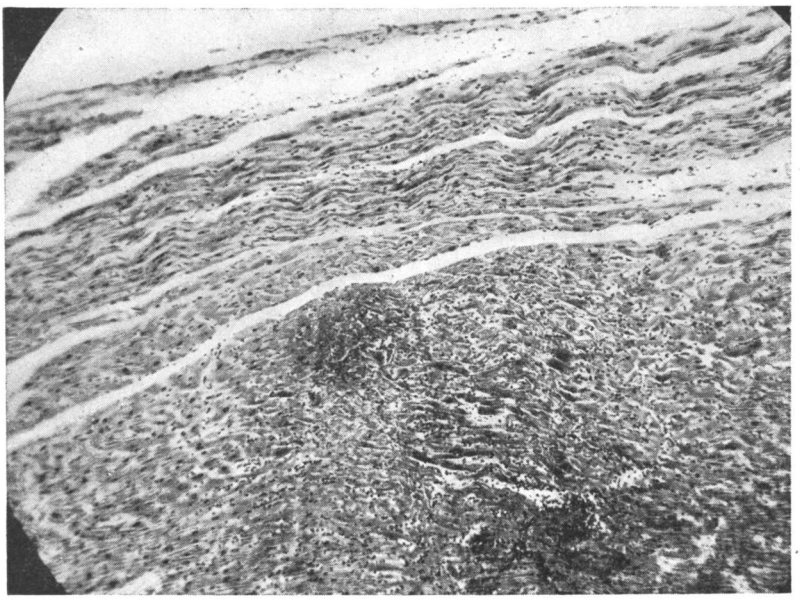

FIG. 2. Subendocardial myocardial infarct. Note the recent infarct and the narrow rim of normal myocardium immediately adjacent to the endocardium.

unconscious. The pulse became weak and rapid, the skin cold and clammy and the patient died 10 minutes later.

Necropsy. An autopsy performed six hours after death disclosed the following findings: Severe generalized arteriosclerosis, nephrosclerosis of the arteriolar variety, moderate hypertrophy of the heart, severe coronary arteriosclerosis with narrowing of branches of the left coronary artery in many places, a recent subendocardial myocardial infarct of the left ventricular wall, edema and hyperemia of the lungs, bilateral hydrothorax, chronic passive hyperemia of liver and spleen, moderate fibrosis of the pancreas, multiple myofibromata of the uterus, multiple minute adenomata of the kidneys and early "cytotoxic" contraction of the suprarenals. There was no gross or microscopic evidence of lesions attributable to syphilis.

The gross description of the heart was as follows: The heart was moderately enlarged, especially in its left portion, and weighed 400 gm. The subepicardial fat

was normal. The chambers and endocardium were normal. The tricuspid, pulmonic, aortic and mitral rings measured respectively: 10.0, 7.2, 7.0, and 8.0 cm. in circumference. The tricuspid and mitral leaflets and the aortic cusps exhibited a slight sclerotic thickening. Within the base of the aortic and the mitral rings were a few calcific areas which extended to the septum membranaceum. The myocardium was red-brown. The right ventricular wall measured up to 0.4 cm., and the left up to 1.7 cm. in thickness. The upper portion of the interventricular septum presented a soft, yellowish-red area on its left ventricular aspect. On section, numerous subendocardial hemorrhages and yellowish brown areas were noted throughout the septum extending into the anterior wall of the left ventricle and into the anterior papillary muscle. The coronary ostia were narrowed by calcific aortic plaques. The right ostium was abnormally low and covered by the aortic cusp. The arteries had a normal distribution. The left one presented a very severe sclerosis and atheromatosis throughout with a marked narrowing of the lumen of its various branches at several points. The right coronary artery showed much less severe sclerosis. Neither recent nor old occlusions were noted in the main branches. The aorta was rigid, calcified throughout.

The microscopic examination of the myocardium showed in the interventricular septum many large areas adjacent to the endocardium of the left ventricle in which the muscle fibers had a smudgy appearance and stained very poorly. Between the muscle fibers were numerous polymorphonuclear leukocytes and lymphocytes and some extravasated red blood corpuscles. The separation between the infarcted and normal muscle was clear cut (figure 2). Areas of recent infarction were also seen in the anterior wall of the left ventricle and in the left anterior papillary muscle.

The infarct was judged to be between 24 and 36 hours old.[1] The remaining myocardium exhibited a moderate degree of interstitial fibrosis and some thickening of the wall of the arteries.

Discussion

This case is interesting because of the presence of an extensive subendocardial infarct involving about two-fifths of the thickness of the left ventricular wall without any recent or old occlusion of the coronary arteries, although the mouths of both coronary arteries were narrowed and the branches of the coronary arteries were sclerotic, of the "pipe-stem" type, presenting numerous narrowings. Physiologically, therefore, there was doubtlessly chronic coronary insufficiency. This insufficiency was probably increased by the presence of arteriosclerotic plaques in the aorta at the mouths of the coronary arteries. A moderate degree of interstitial fibrosis of the myocardium was the only evidence of this chronic coronary insufficiency which must have been present for a number of years. On this basis, and in connection with the presence of an infectious process, the patient developed left heart failure. This, in time, caused slowing of the circulation for which the severely diseased coronary arteries could not compensate and precipitated acute ischemia of the myocardium leading to infarction. Anatomically, this was an extensive confluent subendocardial infarction. The myocardium did not present the picture of focal necrosis which has been frequently described in cases of chronic coronary insufficiency.

In this instance the clinical picture, aside from the absence of pain, was characteristic of myocardial infarct. The electrocardiogram was definitely abnormal, showing a left ventricular preponderance pattern but no changes characteristic of infarct (figure 1).

It is now recognized that concordant S–T elevation in the limb leads or at least the absence of discordant S–T depression is typical of pericarditis due to the involvement of the subepicardial myocardium. Recently the reverse has been suggested as a sign of subendocardial infarction; viz. concordant S–T depression in the limb leads, at least the absence of discordant S–T elevation.[2] On this basis the possibility of a subendocardial infarction might have been considered in this case. The S–T depression in Leads I and II can be considered as indicative of the subendocardial infarction found at necropsy. However, the pattern in this case is not convincing because it might have been produced by left heart strain.

It is amazing that so extensive an infarction could occur in this patient without closure of the artery supplying the area. More interesting is the fact that with so extensive an infarct only the inner layer of the myocardium of the left ventricle was involved. The blood flow on the inner side of the left ventricle is closer to the critical level at which infarction occurs than that of the outer side. This lends support to the concept that during systole the extravascular compression of the coronary vessels increases from the epicardial to the endocardial aspect.[3] The threshold for infarction would therefore be reached with less diminution in coronary flow on the inner aspect.

The most common sites of subendocardial infarcts are the interventricular septum and the papillary muscles of the left ventricle. These areas are farthest away from large coronary branches. A large septal branch was observed by Schlesinger only in a small percentage of human hearts.[4, 5] A second factor is the particular exposure of these regions to intraventricular pressure.

Since the exacerbation of the coronary insufficiency was due to failure of the heart, it is possible that therapy directed to improve the failure might have prevented the fatal outcome. Digitalis is a drug which might have accomplished this.

Summary

A case of extensive subendocardial septal myocardial infarction, occurring without occlusion of the coronary arteries, is presented.

The pathogenesis of this unusual condition and the rationale of therapy are briefly discussed.

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