A Case of Myocardial Infarction, Precipitated by Orthostatic Hypotension in the Presence of Essential Hypertension, with the Autopsy

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The orthostatic hypotension in the presence of essential hypertension with the organic changes in the cardiovascular system is only rarely reported.1-5) The remarkable fluctuation of the blood pressure in this may be a precipitation factor of myocardial infarction as it was suggested by Dr. Mainzer.5) The complete postmortem study in this disorder has been reported only in a few cases.1)

In this case the death was caused by myocardial infarction that seemed to be precipitated by the orthostatic hypotension in the presence of essential hypertension and an autopsy was performed.

CASE REPORT

The patient, a 58-year-old farmer, was readmitted to this hospital because of the light headedness and the tightness of the chest for past 1 month in Nov. 5 1964. He had had hypertension for past 25 years. At that time the blood pressure was 180 mm.Hg in the systolic pressure. He was also pointed to have hypertension 10 years ago when he was admitted to this hospital because of the gastric troubles. The antihypertensive therapy was stopped after 3 months of the therapy because he was asymptomatic.

He was admitted 6 months ago because of the light headedness and the cough in May 15, 1964. On the admission the blood pressure was 200/102 mm.Hg. He was not in acute distress and the pulse was 70 per min. and was regular. The heart sound was pure and the cardiac size was normal. No other abnormal physical findings were revealed. The laboratory examinations: Coprology, normal; hematology, RBC 5.65 x 10⁶, WBC 4,700, differential, metamyelocyte 1, band 14, N. 40, E. 4, Mo. 2, L. 18, plasma cell 1; NPN 25 mg./100 ml.; cholesterol 200 mg./100 ml.; Na in the serum 140 mEq./L., K 4.5 mEq./L., chloride 98 mEq./L.; liver function tests, normal; PSP test, 19.4% in 15 min.; urinalysis, normal; Fishberg's concentration test, max. specific gravity 1.028; electrophoresis, normal; regitin test, negative. The electrocardiogram was within normal limit. He was given ismelin after the admission. The blood pressure was reduced, though he became to complain the dizzy spell when he got up to walk. The blood pressure in this occasion

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revealed orthostatic hypotension. Namely the pressure was 190/100 mmHg at the supine position and it was reduced to 122/74 mmHg in the standing position. So ismelin was stopped and the dizzy spell was improved. He was discharged in Aug. 15, 1964.

At home he was improved more and became to be able to work relatively hard at the farm in that autumn. Two weeks before the admission, he developed the tightness of the chest in the morning suddenly. The dull pain continued for about 1 hour. After this initial attack, he became to develop the same symptoms in the succeeding days.

Fig. 1. The postural changes of the blood pressure and the pulse rate. The upper figure shows the change of them in the supine and in the standing positions. The lower figure shows the same changes after bandaging the lower extremities.
So he was readmitted in Nov. 5, 1964, 3 months after the initial discharge.

Family history and past history: Non-contributory.

The physical examination on admission revealed moderately developed and nourished male in no acute distress. The blood pressure was 180/100 mm.Hg in the supine position and the pressure was decreased to 130/60 mm.Hg when he stood up. The ocular fundi was Scheie 1. The cardiac site was normal and the sound pure. The chest and abdomen were normal. Neurologically he was normal.

Laboratory findings: Urinalysis, normal; PSP excretion test, 42% in 15 min.; the concentration and the dilution test of Fishberg normal; NPN, 17 mg./100 ml.; sodium in the serum, 145 mEq./L.; potassium, 4.6 mEq./L.; chloride, 94 mEq./L.; cholesterol, 150 mg./100 ml.; coprology, normal; hematology, RBC 4.79 x 10^6, WBC 4,400 with normal differential, Hgb. 93%; VMA test, negative; the liver function tests, normal; Wassermann's test, negative; the X-ray film of the upper G-I series, normal; the chest X-ray, normal; BMR, -3%; blood sugar at the fasting time, 81 mg./100 ml. From the examinations of the autonomic nervous system, the following results were obtained. Namely the mecholyl test was S-type, atropin test negative, adrenalin test positive and the cold pressure test negative. The tilt table test for the measurement of the blood pressure, the bandage (pres-
sure) test of the lower extremities and the electrocardiographic findings will be described later.

Hospital course: The orthostatic changes of the blood pressure in this case were shown in the Fig. 1. Namely the patient was laid down quietly on the X-ray table for 20 min. When the blood pressure became stable, the table was vertically tilted quickly. Then the blood pressure was lowered to 105/80 mm.Hg. After 10 min. the table was laid down again, and then the blood pressure was elevated again to 200/100 mm.Hg and it became to the same level to the control period. During these procedures the pulse rate was monitored by the electrocardiogram. The pulse rate was 45 per min. before the test, and it was accelerated to 65 per min. on the standing position and it became low again in the supine position. On lying the electrocardiogram (Fig. 2) was within normal limits. On the contrary, however, on standing TV, was elevated and the supraventricular premature beats appeared frequently, though they disappeared again in the supine position. On standing he complained the palpitation and the light headedness. Also he continued to complain the tightness of the chest of the short duration. The Master's test showed (Fig. 3) the bouts of the supraventricular premature beats and the precordial discomfort was relieved by nitroglycerin. Occasionally he developed this discomfort even at rest.

To examine the more accurate mechanism the autonomic nervous system was examined. He was sympatheticotonic. By the adrenalin test (Fig. 4) the blood

![Fig. 3. The electrocardiogram after the two step test.](image-url)
pressure rose to 250 mm Hg in systole and the pulse rate became 150 per min. with the bout of supraventricular premature beats. He complained the anginal precordial pain. So regitin was given and the blood pressure became low and the anginal pain was also decreased in the intensity, but the pain re-appeared after 10 min. Nitroglycerin was given and the pain decreased again after 2 min. After 10 min. of this the pain increased and therefore the patient sat up. So the blood pressure decreased to 150/60 mm Hg and the pain was decreased. After this he remained to the supine position and became to the same condition.

To treat this patient was very difficult. At first we gave only the sedative to him but he was not improved. So the antihypertensive therapy by reserpine was tried. Though after this, naturally, his blood pressure in the supine position was
lowered, and the orthostatic hypotension became to be seen more definitely until the therapy was stopped. To make the bandage at the lower extremities was beneficial for the orthostatic hypotension, though he was untolerable to this therapy. So again he was treated only with the vasodilator and the sedative, and he was ambulatory. But still he continued to complain the tightness of the chest especially when he waked up in the morning that was relieved definitely by nitroglycerin.

In order to determine the fluctuation of the blood pressure in a day, his blood pressure was measured for three days as the Fig. 5, while the patient was absolutely bedridden. The pressure was low in the morning and became higher in the afternoon. It became lower again at night. He developed the severe agonizing precordial pain on the fourth day (Dec. 3) at 5:30 in the morning and the pressure was 200/100 mm.Hg. At this time nitroglycerine was ineffective and the electrocardiogram revealed the acute myocardial necrosis (Fig. 6). The electrocardiographic and the chemical studies of the blood were done serially and these showed the occurrence of myocardial infarction in this occasion. The myocardial infarction was anteroseptal. He died suddenly at the eleventh day after the attack.

**Autopsy Findings**

The heart weighed 450 Gm. The fresh and old subepicardial hemorrhagic infarction at the anterior and the septal wall of the left ventricle of 2 by 3 cm. in size was seen. The coronary sclerosis was remarkably seen especially at the anterior descending collateral artery, though no thrombosis was found. The kidneys showed only the minute scars of the infarction. The aorta showed the moderate atherosclerosis especially at the lower area than the abdominal aorta. The cerebral arteries were only slightly sclerotic considering the age of this patient. No other abnormalities were found.
DISCUSSION

The first question in this case was whether the orthostatic hypotension preceded the essential hypertension or not. The history in this case showed that essential hypertension was pointed 25 years ago already and on the contrary, however, the symptoms of orthostatic hypotension appeared only 6 months prior to the admission. Also the autopsy study of this case revealed that the kidneys were intact and so it might be ruled out that the renal ischemia due to orthostatic hypotension caused the essential hypertension as Dr. Drenick noted in a similar case. So it might be rational that essential hypertension preceded the orthostatic hypotension in this case.

The second question was the pathogenesis of the orthostatic hypotension. In this case both the systolic and the diastolic pressures decreased and the pulse rate, however, increased. These factors showed that he was the asympatheticotonic type in the orthostatic hypotension. In this type it was necessary to rule out the iatrogenic origin in its cause, because we gave ismeline to him. The blood pressure was high persistently after the cessation of ismeline, though it was true that the clinical symptoms of the orthostatic hypotension became manifest by that medication. So that cause would be ruled out. Hypoadrenocorticocytism and hypopituitarism would also be ruled out from the postmortem study. The main problem to be resolved was the neurological disorders in this case. As far as the brain is concerned, it would have played no part in the genesis of this disorder because clinically no special neurologic signs revealed during the admission and by the autopsy data the brain showed no special organic changes with the slight cerebral arteriosclerosis and so this organ would be ruled out from the pathogenesis of this disease. To determine the location of the damage in the peripheral nervous system was very difficult methodically. In this case the pharmacological examinations revealed that the patient was sympathetically constricted by the infusion of the pressor and the sympathetic system responded well. The cold pressor test was positive and so the efferent vasoconstrictor pathway would be intact. This patient showed no sweating in the agonizing anginal pain and so we thought that he might be neurotic. This might show that the post-ganglionic sympathetic nerve supply to the sweating gland would be poor. The tilt table test for the measurement of the blood pressure revealed the decrease of the pressure from 180/100 mm.Hg to 105/80 mm.Hg in the upright position and this would show the deficit in the arteriolar constriction. It would be necessary to determine, adding to the deficiency in the peripheral nervous system, the efficiency of the adrenergic neuro-effector junctions of the systemic arteriolar beds, the sensitivity of the blood vessels to nor-adrenaline and the amount of nor-adrenaline for the
preservation, though it was impossible in this case. The neuroeffector junctions in the hypertensives are deficient and also the arteriolar constrictor reflexes are damaged and poor to the reflexes stimulation in the alteration of the position. In the hypertensives it is shown that the loss of the plasma fluid is remarkable to the extravascular spaces, and this may be called as the functional hemorrhage. In the senile person, moreover, it is reported that the abnormalities of the central and the efferent sympathetic abnormalities may occur. In this case the bandage of the lower extremities improved the blood pressure in the supine position remarkably and so this imply also that the abnormalities of the venous pooling and the reflex venous constriction might be considered. Such a rare occurrence in the pathophysiologic mechanism was reported initially by Dr. Drenick in 1957. In this country 3 cases have been reported already without the autopsy data. In every case of them the true mechanism is unknown. In the cases with autopsy some showed the changes in the central nervous system and other did not. So these points would be remained to be resolved in the future. So it might be able to say that by the postural change and the two step test these were no abnormalities in the ST and T and the premature beats occurred in the supine position and that the postural changes caused the hypoxia in the myocardium.

The arrhythmia also caused the decrease of the coronary flow. Naturally the direct observation of the precipitation of the myocardial infarction by the orthostatic hypotension is impossible because it would not be able to determine the blood pressure at the very onset of the attack. But we could resume that precipitation from the circumstantial evidences in this case. The coronary flow diminishes in a linear fashion to the same as the systemic pressure decreases. The hypertensive with the coronary artery disease develop the anginal attack by the exercise in the hypotension after it. The hypertensive causes the anginal attack when the blood pressure is lowered. This patient showed the anginal discomfort in orthostatic hypotension and also myocardial infarction was developed when he sat up after the absolute bed rest for 3 days in order to determine the blood pressure.

**Summary**

A case with orthostatic hypotension in the presence of essential hypertension was reported. The fluctuation of the blood pressure in this case might precipitate the occurrence of myocardial infarction. The pathophysiologic phenomena in this rare occurrence were discussed with the review of the literature.
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REFERENCES