CASE REPORT

A Case of Acute Myocardial Infarction with Initial ST Segment Depressin

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The electrocardiographic examination is rarely performed at the beginning of myocardial infarction\(^1\). Recently we performed that examination on a diabetic patient on ward and found the ST segment depression initially on the chest leads in acute antero-septal infarction.

In this report such a rare case is presented and the mechanism of ST changes are discussed.

CASE REPORT

The patient was a forty three year old officeman. He had suffered from Trichophytia pompompholyciformis for past several years. In July, 1962, he was admitted to the dermatological ward and Diabetes Mellitus was pointed initially. At that time urinary sugar was 0.5 gm/dl, blood sugar 174 mg/dl before breakfast and total serum cholesterol 256 mg/dl.

Since then, he had been given a strict diet for diabetes but was transferred to the medical ward for the further control of diabetes because he developed the periproct abscess on Oct., 22, 1962. The family history and the past history were not contributory.

Physical examination on admission revealed a well-developed, well-nourished male. The conjunctiva was normal. The blood pressure was 140/80 mmHg. The pulse rate was 84 per minute and was regular, and the radial artery was sclerotic. The heart was of normal size and the cardiac sounds were within normal limits. The chest and the abdomen was normal. There was no peripheral edema and no sensory disturbance of skin, and the patellar reflexes were absent.

The laboratory studies: On admission urinary sugar 30 gm/day in average and the fasting blood glucose 166 mg/dl ; WBC 18,700, RBC. 5.26 Million and Hgb. 16.0 gm/dl ; ESR 49 mm in one Hr. and 80 mm in 2 Hrs. ; total serum cholesterol was 237 mg/dl. Electrolytes in serum, thymol-turbidity test, zinc-sulphate turbidity test, Brom-sulphalein retention test and Phenolsulphophthalein excretion test were normal. The ophthalmological studies revealed angiosclerosis of retinae (1st grade of Scheie) and cataracta incipiens. The chest X-ray examination revealed normal heart. The electrocardiogram was within normal limits except that T \(_1\) was taller than T \(_a\) (Fig.).

Hospital course: After the admission antibiotics, insulin, diet therapy were given and the abscess was incised on Nov. 1. The anal pain was completely relieved on Nov. 8. and urinary glucose decreased to 2 to 3 gm/day, and WBC and ESR became normal. At p.m. 2,00 on Nov. 26 he developed suddenly a severe, squeezing substernal pain just after the bathing, and he was laid down immediately. At that time the pulse rate was 90/min. and was regular and the systolic blood pressure was 108 mmHg. He was given immediately Vitacameraph intramuscularly and 20% glucose 20cc. intravenously, but he was not improved. He was examined at p.m. 2.40 and he was in acute distress and the blood pressure was 120/90 mmHg. The electrocardiogram
A CASE OF ACUTE MYOCARDIAL INFARCTION

Nov. 9  Nov. 26  Nov. 27  Dec. 25
I  PM 2:40  PM 3:40  PM 6:00
II
III
aVR
aVL
aVF
V1
V2
V3
V4
V5
V6

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revealed marked depression of ST segment in V₁₋₃, QS pattern in V₁₋₂, small R wave in V₁₋₄, small R/S ratio in all of the chest leads and the marked clockwise rotation in the chest leads (Fig.). Persantin and phenobarbital were injected. There was depression of ST segment in ECG and so amyl nitrit inhalation was given, but in fail. The chest pain continued further for sometime, but it was relieved gradually in p.m. 3.40. At that time the electrocardiogram showed normal ST pattern and the clockwise rotation in the chest lead was returned slightly. At p.m. 6:00 the chest pain almost disappeared, but the tightness of the chest in left was remained same. The blood pressure was 147/92 mmHg and WBC 16,100, ESR 10-20 mm. The electrocardiogram showed QS pattern in V₁₋₅, slight elevation of ST segment in V₁₋₄ and moderate clockwise rotation in the chest leads (Fig.).

In the next morning he had only a unpleasant feeling slightly in the chest, but the feeling became good in the afternoon. The electrocardiogram showed marked elevation of ST segment in V₁₋₅ (especially V₁₋₂) and, T inversion in lead I, aV₁, and V₂₋₃ (Fig.). The blood pressure fell to 110/80 mmHg, and serum GOT and GPT increased to 339 U. and 61 U. respectively. Thereafter he was treated with persantin, papaverin hydrochloride by mouth and carboxylyase injection intramuscularly. The course was good and laboratory data returned within normal limit, that is, on Dec. 5 GOT 32 U, GPT 28 U, on Dec. 10 WBC was 8000 per cubic mm and ESR raised to 56-68 mm on Dec. 1 and returned to 14-40 mm on Dec. 21. The electrocardiogram remained same except that r in V₁ appeared and T-wave on chest lead became deep (Fig.).

He was discharged on Dec. 27.

DISCUSSION

It was clear that this attack was a myocardial infarction, because there was the continuous severe precordial pain which was not relieved by the amyl nitrit inhalation, high serum GOT and GPT, high ESR, and QS pattern in V₁₋₂ in the electrocardiogram.

Although the inversion of T wave in an overlying epicardial lead is the first electrocardiographic change of the coronary artery ligation, the inversion returns soon and this change were rarely found in the clinical cases, and the large upright T wave was rarely found. The depression of ST segment in an overlying epicardial lead is the next electrocardiographic change, and means the injuring of the underlying musculature. It appears usually within 6 to 24 hours after the attack in the clinical cases, but in Lamb's case the elevation of ST segment appeared soon after the onset of the infarction. It is quite rare that the depression of ST segment appears initially in myocardial infarction.

According to the classic electrocardiographic theory the depression of ST segment is explained by the reciprocity from the injury current in the underlying subendocardium and Q wave is explained by the transmural necrosis.

In this case it was impossible to explain the depression of ST segment by the subendocardial infarction because QS V₁₋₃ was noted. Although there is a report that the depression of the ST segment may be combined with Q wave in subendocardial infarction, there are not a few reports against it. It may be possible to explain according to Prinzmetal that the depression of ST segment is the reciprocal expression of the infarction in opposite side, but in our case the findings of posterior wall infarction could not be seen of lead aVF, II and III. If the high sited posterior wall infarction was present, it was unreasonable that the electrocardiogram showed QS pattern or large S wave in V₁₋₄, because in such occasion the electrocardiogram should show usually large R and small S waves in lead V₁₋₂. Therefore this depression of ST segment could not be thought by the reciprocity from an injury current of the posterior wall infarction.

Recently Prinzmetal et al. reported a variant form of angina pectoris namely angina pectoris with ST elevation and have made the interesting theory about ST segment change in the myocardial anoxia. They emphasized that the subendocardial myocardium had no relation to the electrocardiographic changes, and demonstrated experimentally that moderate myocardial injury would produce the depres-
sion of ST segment with large S wave and small R wave, and severe myocardial injury would produce the elevation of ST segment with large R wave and small S wave\(^{14}\)\(^{15}\), and moreover observed clinically that mild acute myocardial infarction accompanied transiently with ST depression and lowering of the R wave in chest leads\(^{16}\). They concluded from these observations that the deviation of ST segment was related mainly to the alteration of the balance between intra- and extracellular electrolytes\(^{14}\)\(^{15}\)\(^{17}\).

According to their theory, it was suggested that the initial ST segment depression in this patient was due to the mild ischemic injury and it was combined with QS-pattern in \(V_{1-3}\) due to its transmural quality, and thereafter when the injury progressed to severe grade the typical electrocardiographic pattern of anteroseptal infarction with ST-segment elevation became manifest.

**Summary**

We have reported a case of acute anteroseptal infarction with initial ST segment depression and have discussed about the mechanism.

**REFERENCES**


*Japanese Circulation Journal Vol 28, May 1964*